A STUDY ON INCIDENCE AND TYPES OF OTOMYCOSIS IN PATIENTS DIAGNOSED WITH CHRONIC SUPPURATIVE OTITIS MEDIA

Dissertation submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In fulfilment of the regulations required for the award of

M.S.(OTORHINOLARYNGOLOGY)

DEPARTMENT OF ENT

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

THE TAMIL NADU DR M.G.R MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

APRIL2017

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GUIDE : DR S. PALANINATHAN M.S. ENT

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APRIL 2017

CERTIFICATE

This is to certify that the dissertation entitled " INCIDENCE AND TYPES OF OTOMYCOSIS IN PATIENTS DIAGNOSED WITH CHRONIC SUPPURATIVE OTITIS MEDIA" is a bona fide work of **Dr. Arijit Das** PG student, done under the direct guidance and supervision of **Dr S. Palaninathan** M.S in Dept of ENT, PSG Institute of Medical sciences and Research, Coimbatore,

Dr. S.PALANINATHAN

PROFESSOR AND HOD

Dept. of ENT

Dr RAMALINGAM

Principal

PSGIMS & R, Coimbatore.

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DECLARATION

I hereby declare that the thesis entitled “INCIDENCE AND TYPES OF OTOMYOSISIN PATIENTSWITH CHRONIC SUPPURATIVE OTITIS MEDIA" was prepared by me under the direct guidance of and supervision of Professor and HOD of ENT, DR.S. PALANINATHAN M.S. , PSG IMS&R, Coimbatore.

This dissertation is being submitted to the Tamil Nadu DR, MGR Medical University infulfilment of University regulations for the award of M.S. degree in Oto-Rhino -Laryngology .Thisdissertation has not been submitted for the award of any other degree or diploma.

Dr Arijit Das

CERTIFICATION BY THE GUIDE

This is to certify that the thesis titled "**A STUDY OF INCIDENCE AND TYPES OF OTOMYCOSIS IN PATIENTS DIAGNOSED WITH CSOM"** is a bonafide work of **Dr. Arijit Das** done under the direct supervision and guidance of Dept of ENT ,PSG Institute of Medical Sciences & Research , Coimbatore in fulfilment of regulations of Dr. MGR Medical University for the award of M.S. degree in ENT

Dr. S. PALANINATHAN
Professor and HOD of ENT

PSG Institute of Medical Sciences & Research
Institutional Human Ethics Committee

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To
Dr Arijit Das
Postgraduate
Department of E N T
PSG IMS & R
Coimbatore

Ref: Project No. 14/426

Date: October 28, 2015

Dear Dr Arijit Das,

Institutional Human Ethics Committee, PSG IMS&R reviewed and discussed your application dated 08.12.2014 to conduct the research study entitled "*Prevalence and types of otomycosis in chronic suppurative otitis media in PSG IMS&R*" during the IHEC meeting held on 22.12.2014.

The following documents were reviewed and approved:

1. Project Submission form
2. Study protocol (Version 1.1 dated 14.10.2015)
3. Informed consent forms (Version 1.1 dated 20.10.2015)
4. Confidentiality statement
5. Application for waiver of consent
6. Permission letter from concerned Head of the Department
7. Current CVs of Principal investigator, Co-investigators
8. Budget

The following members of the Institutional Human Ethics Committee (IHEC) were present at the meeting held on 22.12.2014 at IHEC Secretariat, PSG IMS & R between 10.00 am and 11.00 am:

Sl. No.	Name of the Member of IHEC	Qualification	Area of Expertise	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
1	Dr. P. Sathyan (Chairperson, IHEC)	DO, DNB	Clinician (Ophthalmology)	Male	No	Yes
2	Dr. S. Bhuvaneshwari (Member-Secretary, IHEC)	MD	Clinical Pharmacology	Female	Yes	Yes
3	Dr. S.Shanthakumari	MD	Pathology, Ethicist	Female	Yes	Yes
4	Mrs P Rama	M Pharm	Non-medical (Pharmacy)	Female	Yes	Yes

The study is approved in its presented form. The decision was arrived at through consensus. Neither PI nor any of proposed study team members were present during the decision making of the IHEC. The IHEC functions in accordance with the ICH-GCP/ICMR/Schedule Y guidelines. The approval is valid until one year from the date of sanction. You may make a written request for renewal / extension of the validity, along with the submission of status report as decided by the IHEC.

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Following points must be noted:

1. IHEC should be informed of the date of initiation of the study
2. Status report of the study should be submitted to the IHEC every 12 months
3. PI and other investigators should co-operate fully with IHEC, who will monitor the trial from time to time
4. At the time of PI's retirement/intention to leave the institute, study responsibility should be transferred to a colleague after obtaining clearance from HOD, Status report, including accounts details should be submitted to IHEC and extramural sponsors
5. In case of any new information or any SAE, which could affect any study, must be informed to IHEC and sponsors. The PI should report SAEs occurred for IHEC approved studies within 7 days of the occurrence of the SAE. If the SAE is 'Death', the IHEC Secretariat will receive the SAE reporting form within 24 hours of the occurrence
6. In the event of any protocol amendments, IHEC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. Alteration in the budgetary status should be clearly indicated and the revised budget form should be submitted
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval
   d. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented
   e. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IHEC and only then can they be implemented
   f. Any deviation-Violation/waiver in the protocol must be informed to the IHEC within the stipulated period for review
7. Final report along with summary of findings and presentations/publications if any on closure of the study should be submitted to IHEC

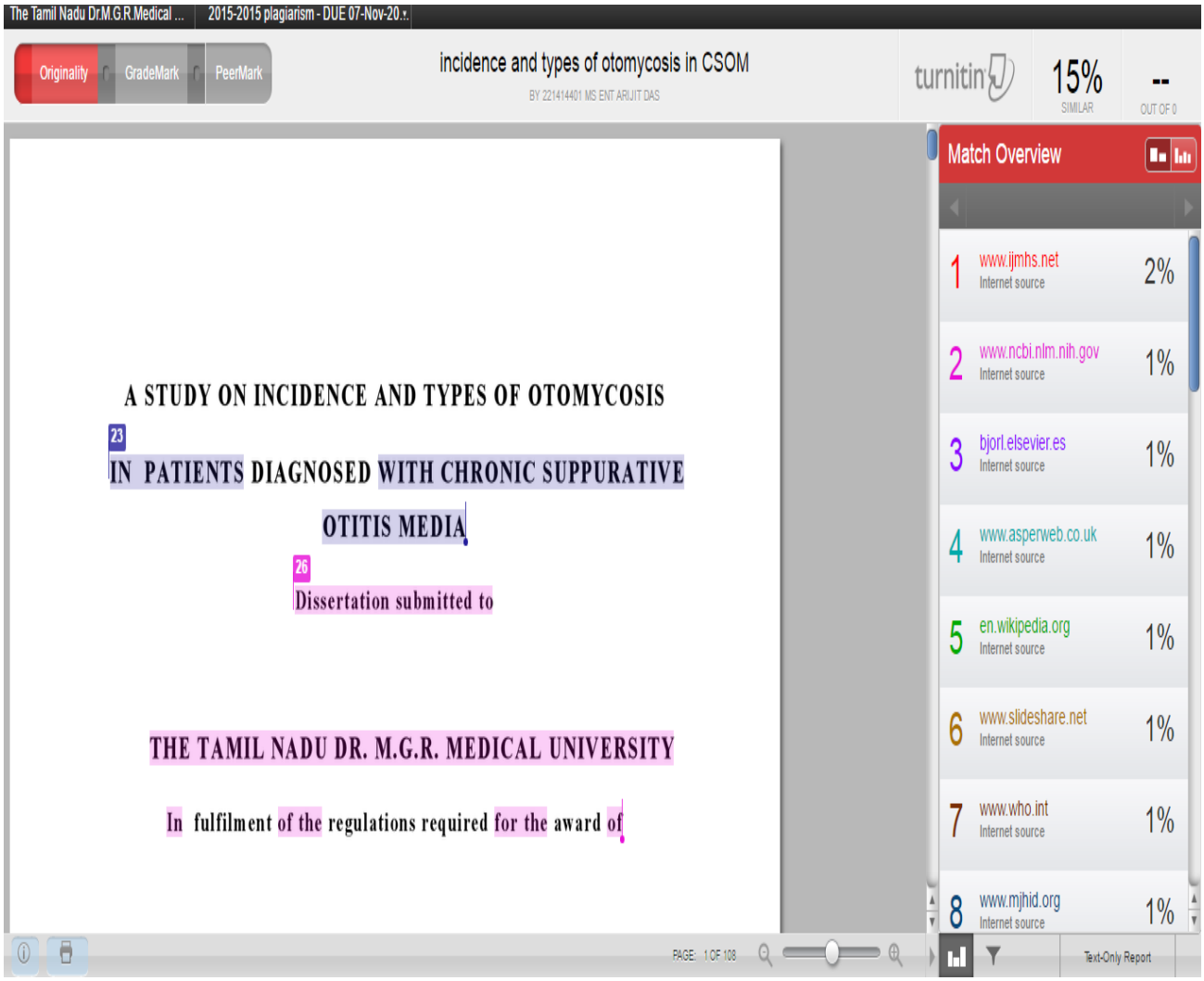
Kindly note this approval is subject to ratification in the forthcoming full board review meeting of the IHEC.

Thanking You,

Yours Sincerely,

SECRETARY
PSG IMS&R
COIMBATORE-641004.
INSTITUTIONAL HUMAN ETHICS COMMITTEE

Dr S Bhuvaneshwari
Member-Secretary
Institutional Human Ethics Committee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HOD of Microbiology, who were always of great help in the effective use of lab facilities , cannot forget the important contribution of assisting me in taking clinical photographs Mr. **Muthukumar** who was very enthusiastic that the cultures should be collected in proper manner and transported soon to the lab.

Words are not enough to congratulate my family, my colleagues **Dr.Dhanasree**, **Dr.Minu** and **Dr Harshvardhan** who helped me whenever required in sample collection.

I can never be thankful enough for valuable assistance obtained from **Dr Damodaran**, Community Medicine Dept for his timely guidance in tabulating the results of the study .

All of ENT Dept staff **Mrs Chitra**, **Mrs Anandi** and ENT general ward sisters for informing me about the status of the sample reports from time to time. I am thankful to **Dr S.Ramalingam**, Dean of PSG IMS &R for having consented to the study and a source of motivation at other times. Finally my heartfelt appreciation to the patients enrolled with their kind consent for the same without whose cooperation this study would not have been possible.

TABLE OF CONTENTS

INTRODUCTION

Otomycosis is a superficial, sub acute, or chronic infection of the outer ear canal, unilateral mostly, that ischaracterized by inflammation, pruritus, and scaling. Otomycosis is a common condition encountered in ENT practice more in chronic or persistent ear infection .It is prevalent more in warm and humid climates.

In Indian subcontinent, otomycosis has been consistently present in many cases presenting with discharging ears.

Predisposing factors such as self-inflicted trauma (use of q-tips to clean itching in ear), failure in the defence mechanism in EAC(epithelial coating changes, changes in pH, quantitative and qualitative changes in ear wax), long term hearing aid /prosthesis, swimming with water trapped in EAC→bacterial infection, abuse of nonspecific broad spectrum antibiotics, steroids, neoplasia and immunological disorders, all of which may render the host prone to the development of otomycosis[2,5,6].

Following clinical exam (otoscopy) it is possible to confirm diagnosis via fungal examination. Aspergillus and Candida spp.are most commonly detected fungi in EAC infections, are opportunistic and usually of varied pathogenicity, being part of the normal microbiota from different body parts[7,8]. Treatment regimens vary

from termination of infection and/or controlling predisposing factors, to local debridement (microaspiration) and/or the use of antimicrobial agents (topic/systemic)[9,10] .

Although otomycosis is a clinical entity throughout the world, there are only a few major studies regarding its true impact and frequency in Brazil [88,89,90].

The present paper is an attempt at assessing the frequency of predominant symptoms and patients suffering previously from CSOM by clinical and mycological diagnosis.

REVIEW OF LITERATURE

Apart from the use of topical antibiotic drops, the moist conditions produced by the discharge alkaline pH , and epithelial debris are also responsible for the secondary fungal infections. Distribution is worldwide with greater incidence in humid , hot and dusty environments of subtropics and tropics[1,2]

Overview of the literature reveals otitis media to be a common medical problem in India[3,4]. According to WHO, defines CSOM as otorrhoea through a perforated TM for atleast 2 wks .As it is accepted that Acute otitis media treated incompletely usually precedes CSOM these variations in transition from acute to chronic imply that consensus has not been established clearly for the transition time .

CSOM should also be understood as a different entity from chronic Otitis media with effusion. Having no active infection or perforation as well as from a chronic TM perforation without presence of active middle ear disease.

Fungi can either be the primary pathogen or be superimposed on bacterial infections or can be secondary pathogen in previously perforated tympanic membrane. Fungal otitis externa can be acute , subacute or chronic caused by filamentous fungi or yeasts affecting

squamous epithelium of external auditory canal . It is mainly characterized by pruritus, ear ache, discharge, hearing loss, aural fullness, and rarely tinnitus.

Multiple factors which usually predispose to otomycosis include immunocompromised host, trauma , ear picking, use of headgear, swimming, use of oils, instrumentation of ear and malnutrition in children[1-7] lack of personal hygiene ,self-cleaning attempts by oil instillation accompanied by indiscriminate use of topical ear drops predispose to otomycosis. Aspergillus is a fungus of the Ascomycetes class that occurs worldwide and may occur as saprophyte , parasite or frank pathogen in man .It is found in moulds on fruits , grain and plants .

Wide spectrum of fungal agents such as Aspergillus, Penicillium, Mucor, Rhizopus, Scopulariopsis, Absidia and Candida are involved, species of Aspergillus and Candida are found to be the most common etiological agents

HISTORICAL PERSPECTIVE:

CSOM first described by Hippocrates as early as 450 B.C., and it still persists to present itself even today as one of the most perplexing universally observed medical problems of childhood and a leading cause of hearing loss [15]. According to WHO survey, 42 million people (older than 3 years) worldwide have hearing loss. The major cause for hearing impairment is otitis media which is second only to common cold as a cause of infection in childhood [16].

It is estimated that about 90% of the people have atleast one episode of otitis media by their second birthday. For children less than fifteen years old, the most frequent diagnosis made in clinical practice for ear discharge is otitis media [17].

Following are few headings to discuss pathogenesis of CSOM :

A) Eustachian tube function—3 important functions of clearance, ventilation and protection which can be impaired by endo/exogenous causes . When a persistent perforation of TM is present either due to a ventilation tube(grommet) or spontaneously , middle ear “gas cushion “ is affected. Resulting in negative ear pressures in middle ear , retrograde movement of nasopharyngeal secretions through ET. Young children are more

prone to such abnormalities due to short, more horizontal floppy orientation of ET. Immunity against organisms by antibody production has been found to be more of local phenomenon than systemic response. The recovery from otitis media is related closely to mucosal immunity in tympanum. Reduced ciliary motility in middle ear and ET mucosa has been implicated for the same.

GERD also has propensity in theory to contribute to ET dysfunction.

Usually a thin pinkish mucosa is associated with a healthy epitympanic processes which becomes thick, edematous maybe with polyps , granulations or cholesteatoma filled middle ear .

Particular note to be made of is infection changes in mastoid and middle ear by exudate rich in fibrin. The primary factor being inflammation to epithelium ,tissue proliferation in growth of granulation tissue into gaps through inflamed epithelium into exudate while normal epithelium attempts to cover the raw areas.

Connective tissue in massive columns form between promontory and ear drum or numerous epithelium lined cavities (cystic appearance) Adhesions follow as this collagenous scar tissue becomes organized .

Keratin and mucus producing cells, have been demonstrated adjacent to each other by Paparella, Bendak, Tos,Friedman). More of this is present in chronic and secretory otitis media. Appearance of cells producing more of mucus(glycoprotein) or keratin is only reaction to minor influence of environment. Nevertheless, these reactions may prove to be the main manifestations of the pathological process.

CSOM is a major reason for acquired hard of hearing in children .Most treatment approaches have been unsatisfactory or or difficult to cure it effectively; eg. Parenteral aminoglycosides are ototoxic potentially requiring long periods of followup and hospitalisation.

With a global burden of more than 330 million people suffering from it at one point of time in their lives, with 60% having significant hearing loss .90% burden of disease is borne by developing countries in S.E. Asia , Africa and Western Pacific regions.

Complications of CSOM:

Chronic mastoiditis →Middle ear , mastoid cavity erosion lead to Facial Nerve injury/paralysis, lateral sinus thrombophlebitis, meningitis, labyrinthitis, brain abscess.

Bottle feeding in infants , attending congested centres like day care centres , passive smoking are some of the risk factors for development of otitis media.

Topical quinolones are more effective than other antibiotics (ofloxacin /ciprofloxacin) than IM gentamycin , neomycin-polymyxin for otorrhoea.

Combined topical and systemic antibiotics are no better than topical antibiotics use alone.

CSOM amenable to medical management is mainly to control infection , eliminate otorrhoea , and improvement of hearing by eventual healing of perforation as long term goals.

Gavarret and Andral in first described fungal infections in external auditory canal in 1843,following which Virchow suggested the term ‘otomycosis’. Wolf described the relationship of various fungi to this condition. Pacini in 1851 first described preparation tomanage otomycosis . Cleansing and drying were postulated as essential to management to aid in symptomatic management but doubt of need for any additional measures necessary remained controversial. In other studies, Lakshmipathi, Geaney and Murthy showed that all cases observed wereaffected by Aspergillus or Candida species [6,7] .

There is abundance of fungi in vegetable material , soil which following drying up in tropical climates and windblown as small fungal spores , which is evident with increase in infection rates with the onset of rains, (relative humidity may be upto >80%.)

The prominent role A. niger has in otomycosis was further exemplified in the 60's and 70's (Damato et al, 1964; Bezjak, 1970) ,the formerstrained on cleansing and drying to be beneficial in reducing recurrence to huge extent with emphasis on the role tolnaftate could have in its management .

Stern et al suggested were also proponents for the idea of meticulous drying , cleansing. Otomycosis originally has been suggested as a fungal infection of the EAC.Expansion of the terms meaning to include fungal infection upto middle ear and mastoid cavities was suggested by ***Paulose et al.***).

Haruna et al in 1994 added to histopathological observations in cases

ANATOMY

Development of external ear :

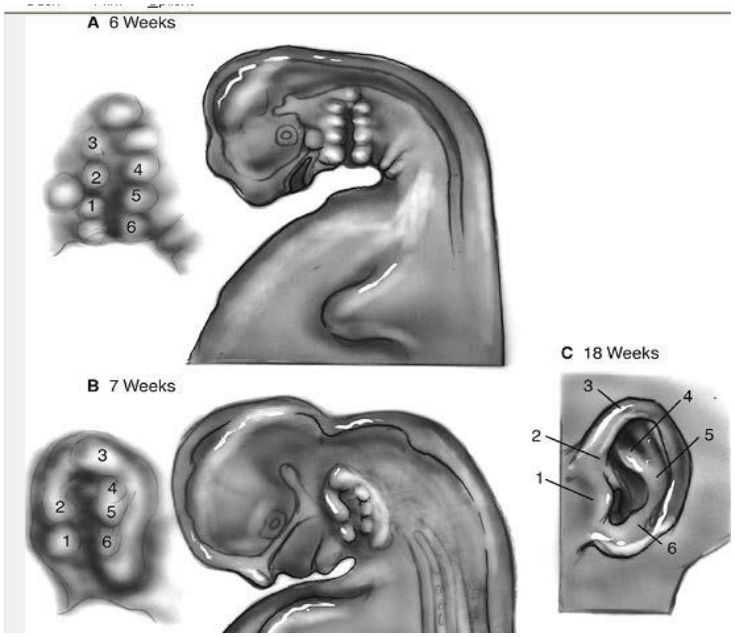
The otic placode is the first hint of a future ear and is present during the third week of intrauterine growth. The auricle forms

frommesoderm of the 1st and 2nd pharyngeal arches; its growth occurs via development of six hillocks of His around 5thweek . The following six structures evolve from these hillocks:

1. tragus, 2. helix, 3. helical crus,

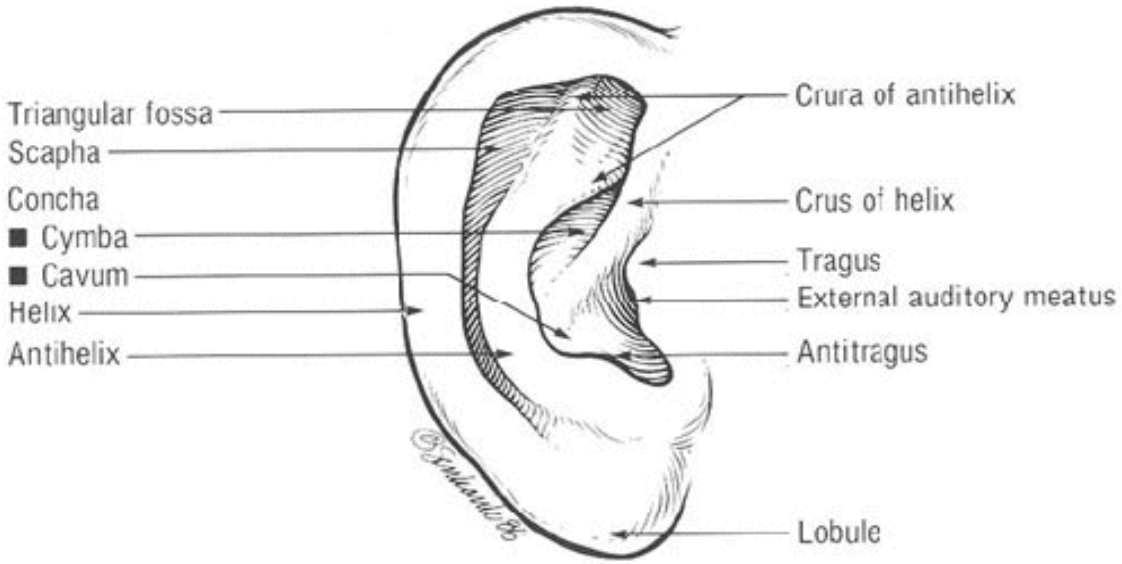
4.antitragus, 5. antihelix and 6. lobule.

The external ear is composed of the auricle and external auditory canal having subcutaneous tissue and elastic cartilage (mesoderm) , protected by skin and adnexa. Arising from dorsal part of 1st branchial cleft between 1st and 2nd branchial archesby 8th wk primary External auditory meatus (future lateral 1/3rd of EAC) is formed surrounding mesoderm forms the cartilage around. Endoderm of 1st pouch in contact with cleft allows mesoderm to grow between the layers with most medial cells of the ectodermal meatal plug forms outer layer of TM.



3 hillocks in the 1st arch , and remaining (hillocks 4, 5, and 6) from 2nd arch on both sides of the 1st cleft. Final fusion occurs by 12th week .

Fat is present in lobule but no cartilage . EAC is usually around 24 mm long with 1-2 ml volume.Outer 1/3rd canal is made of fibrocartilage, whereas the medial 2/3rd is bony.



External auditory meatus

it is not possible with this view alone to understand the 1st and 2nd bends or the A-P orientation differences in canal. As it does not let us visualise the course above and below (as a result of upward prominence of bone just after the bony-cartilaginous junction .hence the constriction naturally formed called ‘isthmus’. Distinct

angulations present incanal divide it into 3 sections outer 1st and 2nd cartilaginous and inner 3rd bony part.

the horizontal plane occur at the first and second bends of the canal. The angulation at 1stis termed as “conchomeatal angle .An appropriate name for the angle formed at second bend is “cartilaginousbony angle (CB). Water and debris have easy chance of being trapped easily in lower angle of Tympanic membrane referred to as sulcus.

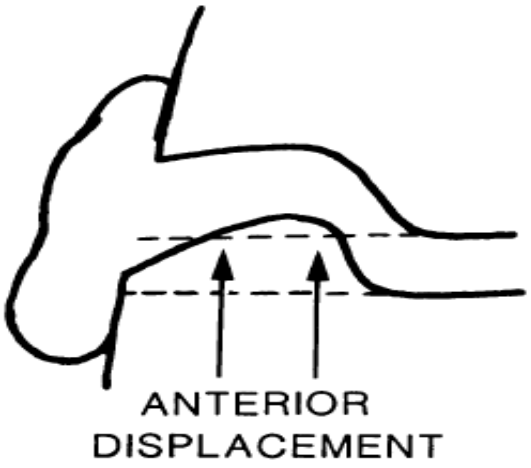
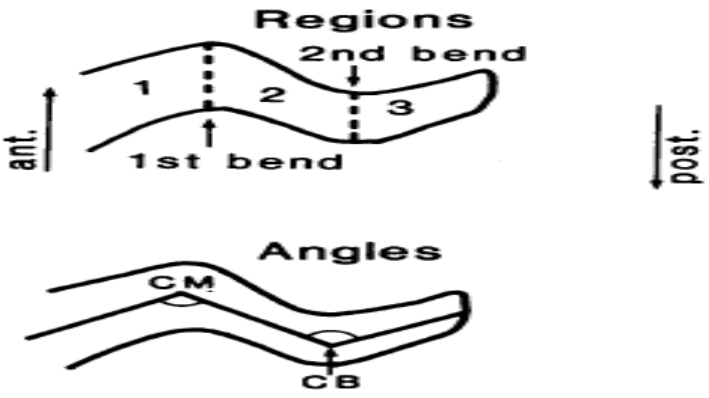
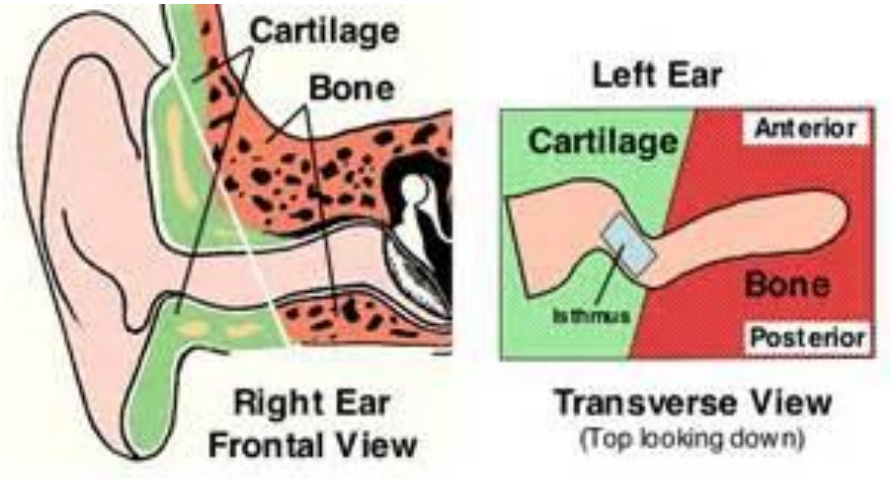


Figure 7 Axial view of a RT external auditory meatus, viewed from below. Note that anterior displacement of cartilaginous canal results in flaring beyond the second bend.





Concha extends to continue as cartilaginous part while the bony portion is developed from squamous and tympanic portions of the temporalbone. Hence ,the external auditory canal which is initially a rather straight tube early in life assumes “S” shaped orientation in adulthood with length being superiorly 2.5cm approx.. and inferiorly 3.0cm.

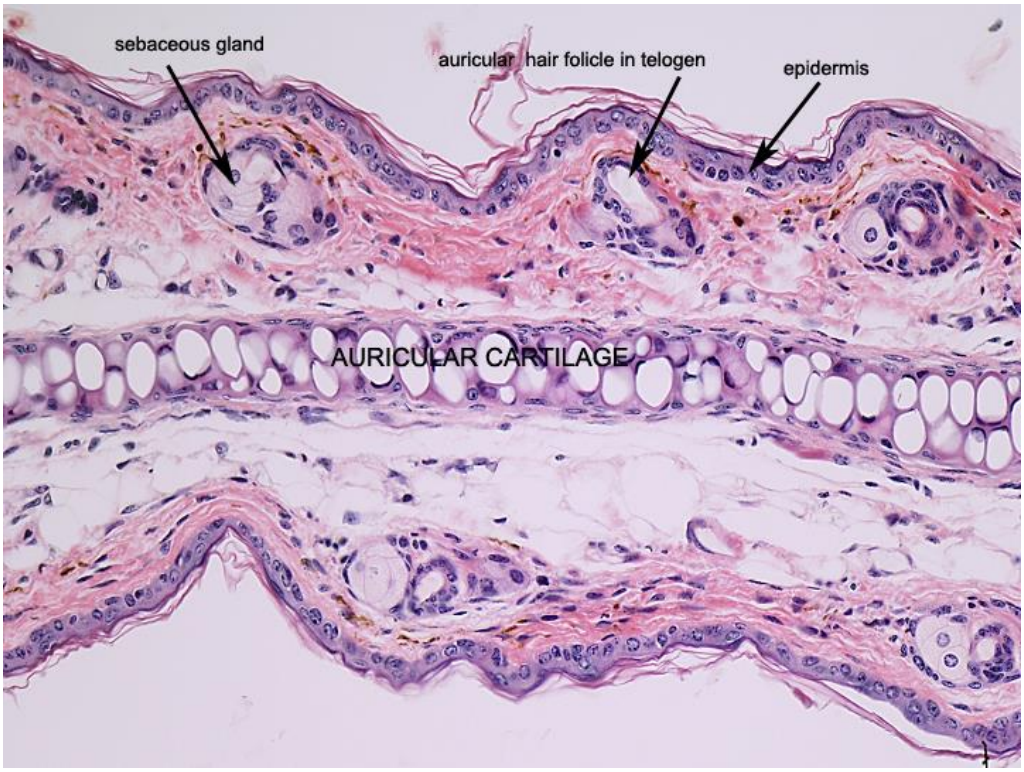
SKIN

Stratified squamous epithelium lines the EAC which continues with the pinna skin and the epithelial lining of the TM. hair follicles, sebaceous and ceruminous glands are found in subcutaneous layer the cartilaginous canal < 1 mm in thickness. The bony canal lacks subcutaneous elements and is merely 0.2 mm thick. Coronal section of the ear canal shows magnified view of skin of the cartilaginous canal. Ceruminous glands (Modified type of apocrine sweat) excrete cerumen which primarily has antibacterial properties , and has

slightly acidic pH and protects from EAC maceration to jointly contribute to unsuitable environment to fungi .But cerumen if excessively viscous may also obstruct, retain debris and water, infection may supervene.

Keratin is known for absorbing moisture forming excellent medium for bacterial growth. With these defences hampered, external otitis may result giving scanty white thick discharge , maybe blood tinged with granulation tissue and /or fuffy to off-white otorrhoea with small white or black conidiophores on hyphae .In case of thick secretions, with crusting positive , H_20_2or antibiotic topical drops will soften them .Unless TM can be fully observed, it is not advisable to flush out /syringe out ear contents avoiding potentially ossicular disruption /hearing loss, giddiness and tinnitus.

In case of edematous /narrow canal wall, cotton wick can be carefully inserted for applying topical medications and facilitate drainage.



Pathogenicity of fungi is attributed to minor intradermal abscesses or abrasions in EAC and TM with hemorrhagic granulations resulting in thrombosis of blood vessels situated adjacent and hence prone to avascular necrosis and finally perforation of tympanic membrane.

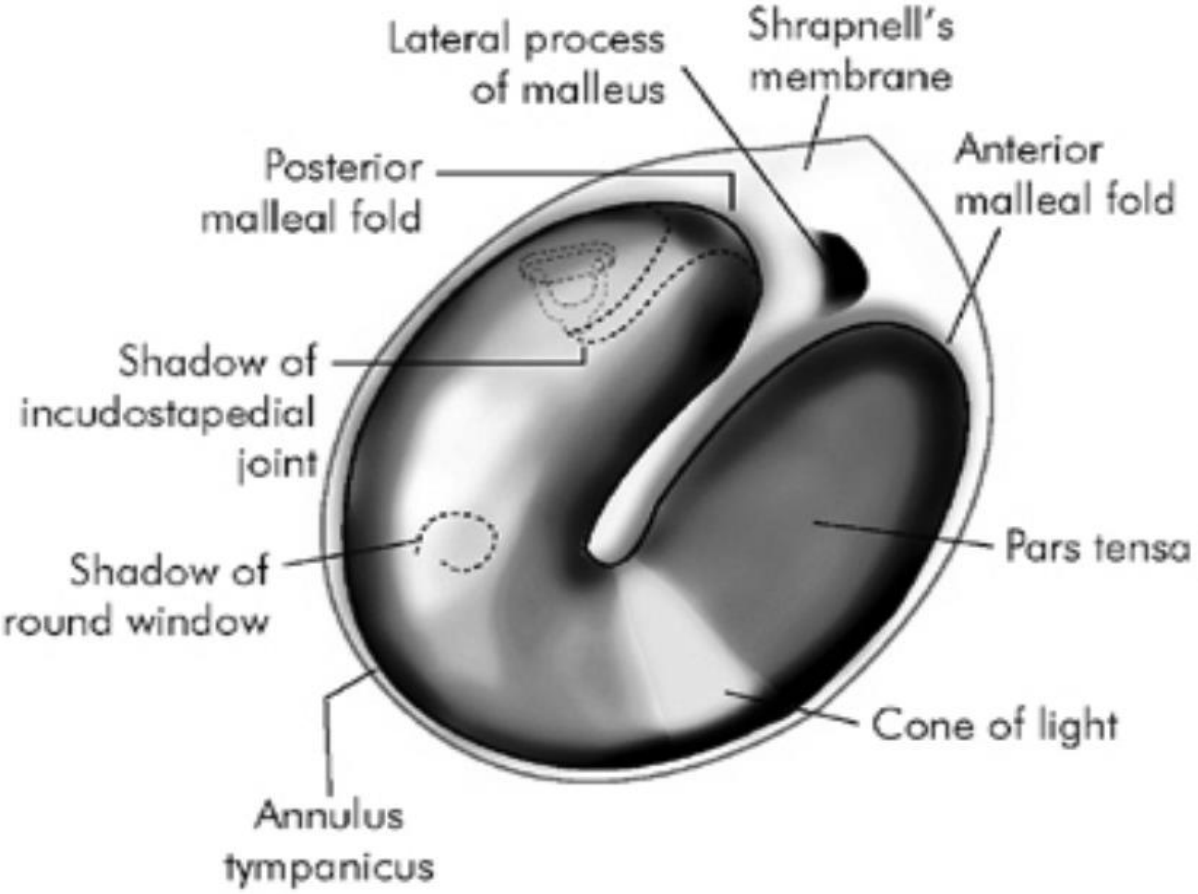
Tympanic membrane :

TM is a translucent, smooth, pearly-white membrane with avg. thickness of 0.074 mm .elliptical and approximately 10 mm high and 8 mm wide weighing approximately 14 mg .slanted at an angle of around 55 ° to floor ,

The TM is slightly concave , being displaced inwardly by about 2mm except at umbo where there is an outward protrusion due to inferior tip of manubrium of malleus.

The outer edge of TM is a fibrocartilaginous ring or annulus (annular ligament), embedded in a groove in tympanic bone called tympanic sulcus which is deficient superiorly at notch of Rivinus.

TM is thickest anterosuperiorly and inferiorly near the annulus (.09 mm) and most thin in posterosuperior quadrant (.054 mm). considering its dimensions , it is a uniquely resilient structure due to its layered architecture .



- **3 layers of pars tensa: outer epithelial layer** from ectoderm of 1st visceral cleft**,**
- **middle fibrous layer** from mesoderm between 1st visceral cleft and tubotympanic recess **& inner mucosal**layer from a part of the recess
- **Fibrous layer disorganized in pars flaccida**

Outer cutaneous layer differentiable into external squamous and deeper cuboidal epithelium. Outward migration of outer epithelial layer begins at umbo continues outward through EAC (rate of this centrifugal at eardrum around 0.05 mm /day).

Middle fibrous layer maybe subdivided into outer Radial fibres (giving the tent like shape of TM)and inner layer of circular (circumferential) fibres giving it flexibility ..Middle ear cleft consists of middle ear cavity , mastoid air cells , Eustachian tube, aditu and mastoid antrum. It is an irregular space containing the ossicles malleus , incus and stapes .

Middle ear :maybe divided into

1. **Epitympanum:**lies above level of short process of malleus .Separated from hypotympanumand mesotympanum by mucosal membranes and folds.laterally bounded by pars flaccida superior to anterior and posterior malleolar folds ,lower part of wedge shaped attic wall called scutum .

Medial : Bone superior to facial nerve(tympanic part)

Contents : head of the malleus , body of the incus

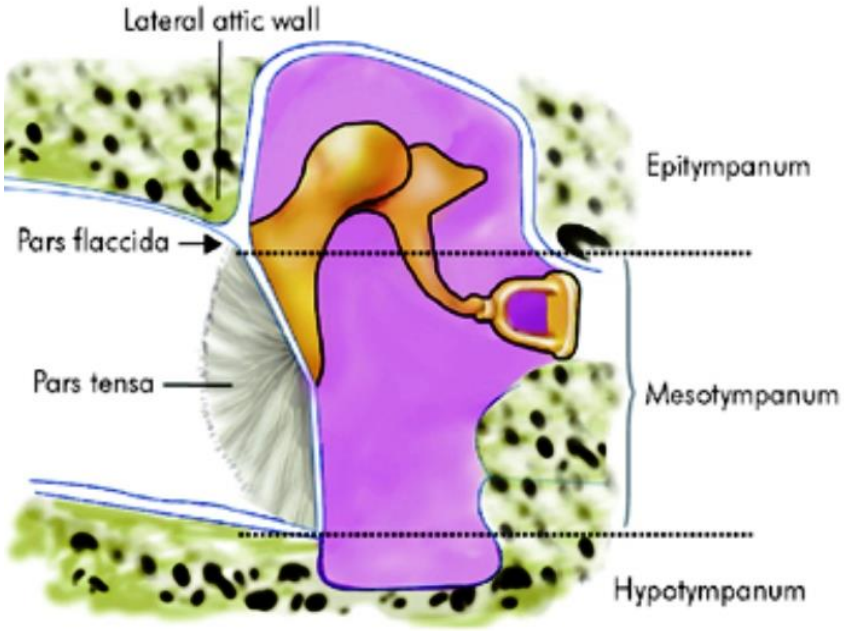
2. **Mesotympanum:** Middle ear medial to pars tensa & tympanic annulus

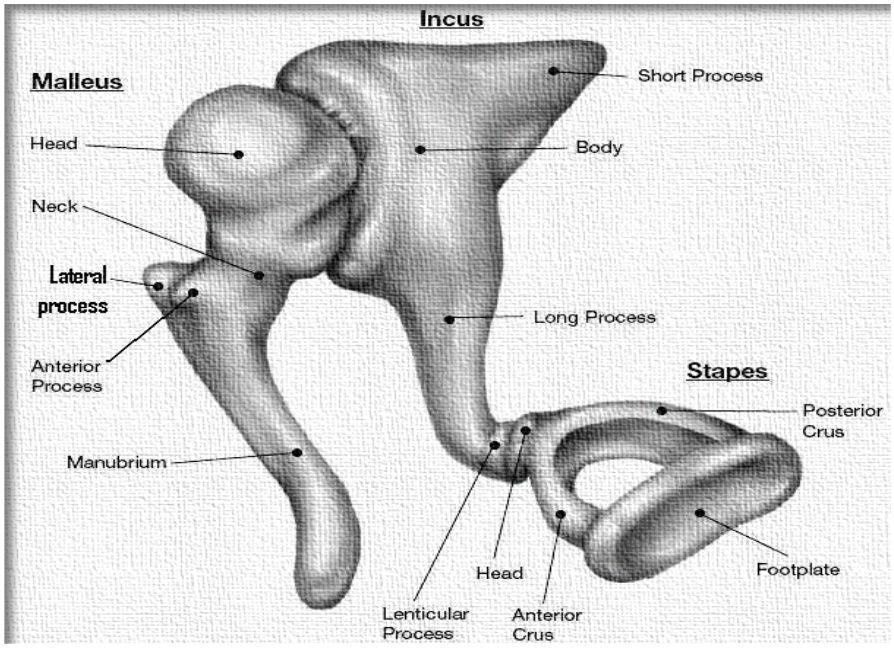
   2 recesses extending backward difficult to visualize often are sinus tympani , facial recess – very common locations for cholesteatoma .Contains long processes of malleus, incus, stapes, oval and round window, CN VII and partly stapedius and tensor tympani muscles.

3. **Hypotympanum:** Middle ear cavity below level of bony tympanic annulus

4. . **Anterior tympanum or Pro-tympanum:** Middle ear cavity in front of bony tympanic annulus

5.**Posterior tympanum:** Middle ear cavity behind bony tympanic annulus





Malleus handle connects with TM loosely to short process via small cartilaginous insert on its end .Stability is achieved at umbo where collagen fibres of TM converge .

Malleus head and body of Incus articulate via saddle-shaped joint

Incus lenticular apophysis connects with head of stapes in a shallowjoint (ball and socket cartilaginous type), stapes footplate having a fibrous attachment with the oval window rim with helpof annular ligament. A single layer of epithelium line all ossicles middle ear cavity.

Tensor tympani muscle from cartilaginous bony wall of Eustachian tube turns almost 90 ° at processus cohleariformis continue to upper part of manubrium. Tensor tympani gets supplied by branch of Mandibular nerve (CN V).

It contracts when the sound stimulation is strong, sudden and threatening enough to produce a "startle reaction".

Arising below from pyramid in posterior wall of middle ear, stapedius muscle(smallest striated muscle in body)continues and inserts into neck of stapes.

Contraction of stapedius muscle causes fixation of stapes helping to reduce transmission up to 30 dB for lower frequencies < 1-2 KHz.

Functions of acoustic reflex:

1. Protection of inner ear from damage due to excess noise.

<u>vascular, lymphatic and nerve supply to the ear.</u>

Sensory innervation :

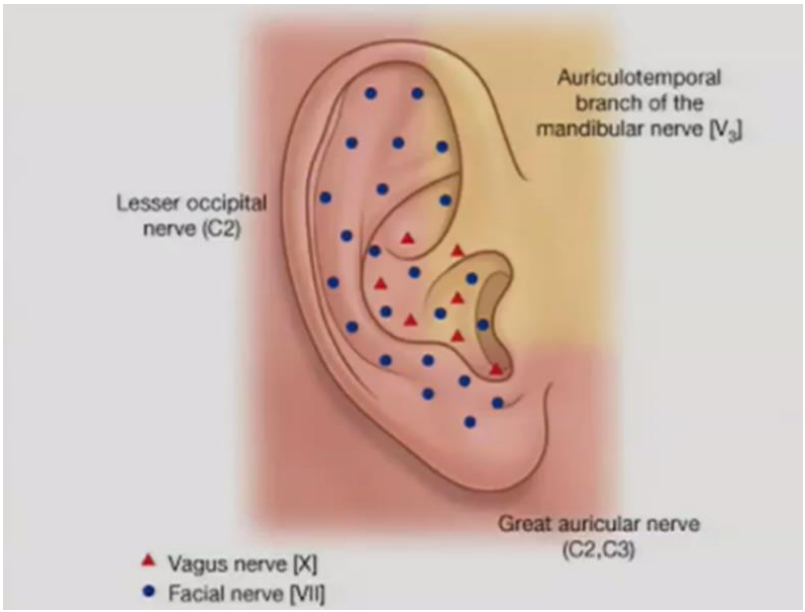
1) CN V (the auriculo temporal nerve for anteromedial and anterosuperior pinna),

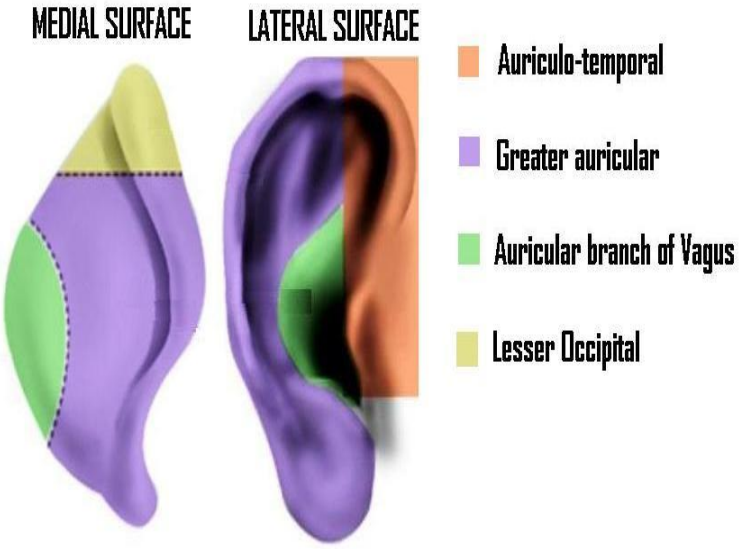
2) greater auricular nerve (derived from C2,3)

. supplies postero lateral , postero medial, and inferior auricle

3) CN X (Arnold's nerve supplies the concha),

4) CN VII (supplying sensory fibres to EAC in posterior and inferior pinna),





Blood supply :

1) ECA(external carotid artery) posterior auricular Artery.
   Superficial temporal A with Occipital artery.

2) Venous drainage by the, external jugular, posterior auricular, superficial temporal, and retromandibular veins.

Lymphatics drain to the parotid LNs anteriorly to cervical LNs. posteriorly

EXTERNAL AUDITORY MEATUS : The EAC consists of Outer 1/3RD cartilaginous part. inner 2/3rd bony part connected with each other termed as bony -cartilaginous junction .

MIDDLE EAR :

Various names have been used to air filled anatomic middle ear spaces anterior malleus (head). Proctor[80], discussed development of these spaces (1964) mentioned about an anterior extension of the attic, (supratubal recess). Trillsch and Wigand(81) described it as sinu epitympani.

Terms like supratubal recess, anterior attic recess, recessus protympanicum and geniculate sinus have been used synonymously. Between 3rd and 7th fetal months, there is gradual absorption of the gelatinous tissue in middle ear cleft. Simultaneously, the primitive tympanic cavity forms in growth of endothelium lined from the Eustachian tube into the cleft.

The middle ear spaces are hence developed from 4 sacs /pouches (saccus medius, anticus, superior and posticus) as outgrowths from eustachian tube.

Saccus medius→attic divide later into anterior , medial and posterior saccule.

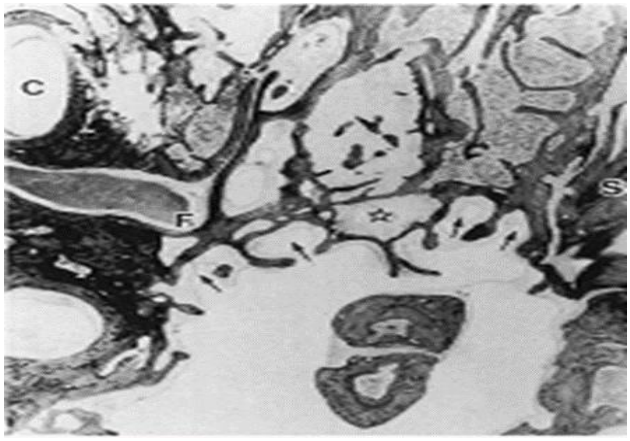
Saccus anticus/ anterior scaccule of sacccus medius →anterior epitympanum. The slower developing saccus anticus meets the

anterior saccule at level of the semicanal for tensor tympani, hence giving rise to horizontal tensor tympani fold.

The space thus formed above the tensor fold and anterior to the tensor tendon is the anterior attic compartment.

Below is a picture of microscopic view of recesses during development :

Air cell like spaces :supratubal recess surrounded by petrosal cells _arrows) C : cochlea ,L :Lateral SCC F: Facial Nerve



Clinical picture :

Considerable difference exists in clinical appearance of aspergillosis and candidiasis of the ear . Aspergillosis presents usuallyas moist inflammation changes in deep ear canal filled with large keratin debris having wet tissue-paper appearance. Candida causes more maceration and edematous in ear canal filling up lumen with curd like debris.

Otomycosis may even lead to TM perforations AND invasion to middle ear. to maintain clinical scenarios documentation and standardization ,it becomes imperative for otolaryngologist to define otomycosis as primary or secondary.

1) **Primary otomycosis** :- Otomycosis in EAC an immunocompetent person in presence of intact TM with no other external or middle ear pathology.
   A. Absence of otitis externa. at time of presentation (canal inflammation / stenosis of the EAC
   B. Presence of signs of otitis externa. (due to otomycosis and not vice versa.

2)Secondary otomycosis. When preexisssting otomycosis is present in the EAC alongwith existing history of otitis media/externa and/or h/o postoperative ears or trauma, with fungal infection in other parts

.(a)Immunocompromised absent . No comorbidities like HIV, DM, or granulomatous diseases

.(b)Immunocompromise is Present as detected by lab studies

RELEVANT MYCOLOGY

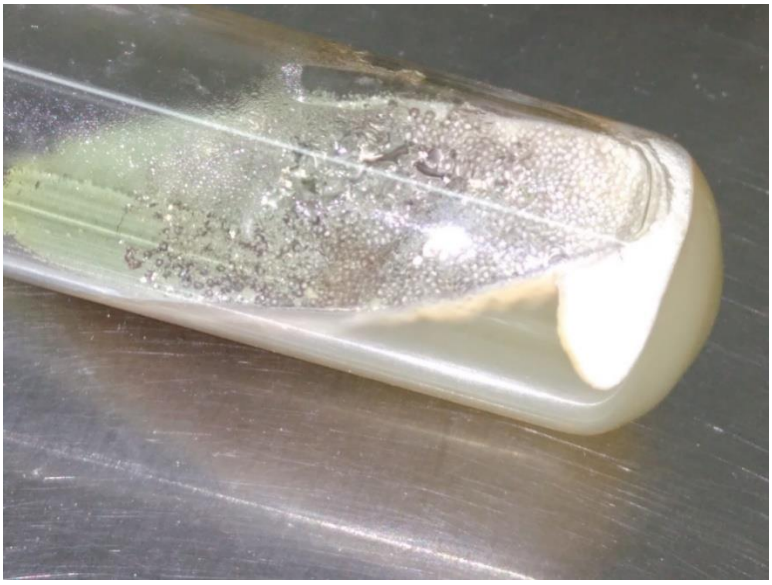
Colonies are usually in shades of green to white sometimes comprising a thin felt of conidiophores. Under microsope , chains of conidia(single celled maybe divergent or in columns , cylindrical , ellipsoid, globose).Conidiophores are smooth – rough walled hyaline with phialides flask shaped , narrow pointed apical part ,a cylindrical basal part and distinct neck.

Aspergillus invades multiple organ systems of body.Naosorbital. Endocardial .Cutaneous . Central nervous system Pulmonary (Allergic bronchopulmonary aspergillosis : fungal ball , invasive pulmonary aspergillosis)

Asp. fumigatus colony resembles cottony , powdery or velvety whitish initially changing to dark greenish to gray /tan growing best at 45degree Celsius uniseriate club shaped vesicles(30-50μ diameter) with phialides ,conidiophores long (300 micrometre) conidia on distal half arising in chains tending to sweep towards central axis .On microscopy , uniseriate phialides

A typical colony of Aspergillus fumigatus in our Microbiology lab.

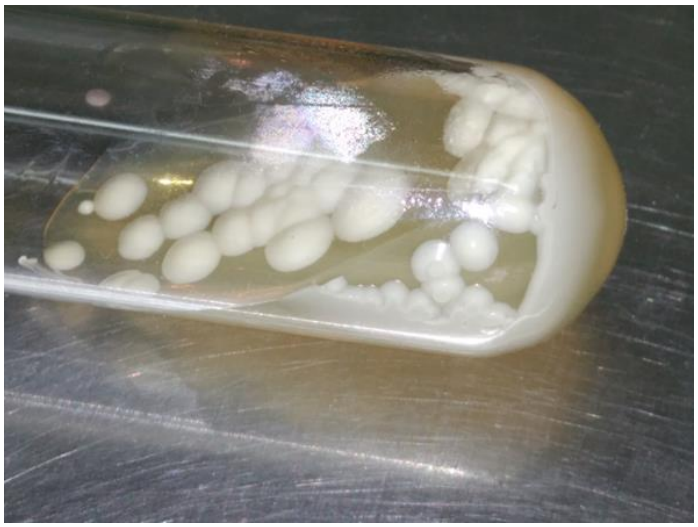
Apergillus flavus is commonly associated with aflatoxins.Rough, spiny conidiophores with brown to yellow to green velvet colonies, uni and biseriate phialides in all directions cover entire vesicle.



Aspergillus flavus colonies in Test tube and culture plate respectively.

Candida :

Yeasts are unicellular budding organisms.(do not produce mycelia) usually forming visible colonies with 48 hours in culture plates .Moist , soft growth of these polymorphic variety of yeasts (hyphae, yeast cells , pseudohyphae .



A culture tube showing Candida colonies grown in our lab(cream coloured smooth surfaced , waxy).

Over 20 species of Candida(commonly Candida albicans, C. parapsilosis, C. glabrata, C. tropicalis) are known to infect human living on skin ,mucous membranes. Secreted Proteases aid in virulence of Candida spp in invasion of keratin protective layer to initiate EAC infection to further result in increased vascular permeability , congestion , raised temperature and decreased pH.Adherence of yeast to skin cells is supposedly facilitated by acid protease proportions .

Dermatophytes: group of closely related fungi causing superficial infections 10-20% (keratinophilic) .Main three genera Microsporum ,Trichophytonand Epidermophyton .It is not always possible by usual KOH mount microscopy(low specificity) and fungal culture(low sensitivity) to make confirmatory diagnosis in these infections .Zoophilic or anthrophilic in nature ,they are well suited to life on skin eliciting minimum inflammatory reaction.Polymerase chain reaction with usage of primers targeting dermatophyte specific sequences of chitin synthase 1 gene maybe considered gold standard . Hints at diagnosis

- *Epidermophyton*: absence of microconidia ; clubshaped macrconidia with smooth thin walls.
- *Microsporum*: multicelled macroconidia with rough, thick walls.
- *Trichophyton*: macroconidia mostly absent .If present, pencil-shaped with smooth , thin walls. 1-2 μ microconidia considered abundant

In our case we had 10% cases of miscellaneous fungi including Derrmatophytes .The most appropriate treatment for the same is important as for example ; Griseofulvin is only effective in dermatophytic infections (no action to Candida spp. and other moulds .Terbinafine is fungistatic against Candida but fungicidal against Dermatophytes .

PATHOGENESIS AND PREDISPOSING FACTORS

Spread of otomycosis can be linked to histology and functioning of EAC. Stratified keratinized squamous epithelium along external side of TM lines this 2.4 cm long, 9 mm wide canal. Medial to isthmus at interior tympanic recess, remains of keratin and cerumen tends to accumulate and it's difficult area to clean. Cerumen is bacteriostatic and antifungal to some extent contains lipids (50-

70%), proteins, lysozymes ,free amino acids ,immunogloblins and PUFAs. These fatty acids in unbroken skin possibly inhibit growth of bacteria.Hydrophobic nature of cerumen makes canal wall surface impermeable avoiding damage to epitheliuma and maceration .

Cerumen also protects against intrusion by insects .EAC harbours normally microorganisms like Staph. epidermidis, Corynebacterium sp, cocci (Staph. aureus, Streptococcus, Gram-negative bacilli (Pseudomonas aeruginosa, H. influenza, Moraxella etc) and as Aspergillus , Candida sp. This balance of commensal non pathogenicorganisms exists in healthy non immunodeficient people. Majorrisk factors which may cause saprophytic fungi to behave as pathogen : -

→ Increasing skin pH level in EAC (excessive bathing). Swimmingregularly without adequate ear protection can predispose to otomycosis. In addition, cerumen has been speculated to be supportive for fungal growth. Consensus on this theory is not generally achieved .

➔ Environmental (heat, humidity) – epithelial alterations .

- Systemic factors (corticosteroids, reduced immunity, morbidity of debilitating conditions, tumours.Overzealous use of Ofloxacin

otic drops may even predispose to otomycosis. (.Jackman et al (2005)

.Presence of longstanding CSOM , bacterial otitis externa and manipulation in post op mastoid cavities are easy targets for fungal contamination providing disrupted skin as a good medium for growth of fungi.

Epithelial damage reduces apocrine and cerumen glands capacity to excrete the protective secretions which modifies EAC environment making it ideal for unusual microorganisms(reduced pH 3-4) .

- Social habits and conditions in females wearing traditional head covers (veils)have been reported to influence chances of otomycosis. Humidity in ear canal increases to create ideal pH for fungal spread.

- protective acid/lipid balance of the ear is lost.

- Dermatomycosis is also a factor on otomycosis specially by autoinoculation

Bacterial otitis externa and clinical otomycosis often have indistinguishable symptoms. However pruritus mostly frequent in fungal infections, associated with ear discomfort, otalgia, ear

discharge, feeling of ear blocking sensation , hard of hearing and tinnitus .

Usually presenting symptomatically as unilateral only rarely B/Las has been documented .

A flavus, niger fumigatus, Penicillium, Absidia, Rhizopus and Scopulariopsis are the commonly encountered agents with Candida sp, especially C. albicans being part of normal human flora with propensity to cause otomycosis.[5,9]

Aspergillus has in past caused even invasive (malignant) otitis externa with local extension beyond bone and cartilage, proving severe ,potentially fatal. This is more commonly seen in immunodeficiency, diabetes& uncontrolled cases. Aspergillus fumigatus is more responsible for such morbidity when compared to Aspergillus niger. Aspergillus has rare chance of causing tympanomastoiditis in some immunocompetent people also.

Management of *Aspergillus* sp. induced Invasive otitis externa classically is extensive surgical debridement, long-term antifungal therapy with Itraconazole/Amphotericin B. Despite such precautions, this condition can be associated with significant morbidity and mortality, due to late diagnosis, comorbidities

present already in patient [91]. Treatment failure due to the side effects of amphotericin B, may require suboptimal therapy and interruption/ decrease of antifungal agents. Currently, voriconazole is considered for first line therapy in such scenarios.(high intrinsic anti-Aspergillus action and well tolerated with good distribution in systemic than Amphotericin B).

CSOM directly may impact hearing in patients CHL and SNHL).It also affects development of a child (WHO,1998). Prime risk factors for CSOM being Upper respiratory tract infections ,necrotizing otitis media, otitis media with effusion and poor living standards with lack of access to proper health care.

In case of accompanying concurrent TM perforation with severe otalgia, acute suppurative otitis media possibly by Aspergillus and/or other fungi has to be considered.

Otoscopy may show mycelia, confirming the diagnosis. Erythematous EAC with fungal debris with or without ear discharge greenish , black fuzzy OR white growth mixed with debris filling up the EAC and classical wet ‘blotting paper’ appearance with white mycelium maybe found .

Slight conductive deafness can be present due to mechanical block in the EAC . Local Hyperemia and minor bleed locally is expected in severe inflammation.

Confirmation :

Confirmation is to be done by initial screening test for identification of fungal elements in a KOH mount followed by fungal culture in case of confirmation.

The characteristic examination resembles that of common moulds, with visible, delicate hyphae and spores (conidiophores) being seen in Aspergillus.

black headed filamentous growth (wet newspaper appearance) in Aspergillus niger, white colonies (cotton wool or wet blotting paper appearance soft caseous material with epithelia debris) in Candida albicans, blue or green colonies in Aspergillus fumigatus & yellow colonies in Aspergillus flavus,

Candida, a yeast, often forms mycelia mats with white character when it’s mixed with cerumen they appear yellowish.

Candidal infections can be more difficult to detect clinically because of its lack of characteristic appearance like Aspergillus and not responding to aural antimicrobial. Otomycosis attributed to Candida is often identified by culture data.

Hematoxylin and eosin staining is useful but not fully able to delineate fungal cell wall. Silver staining is more sensitive for staining but colours cells too dark.

More specific but less sensitive method is PAS (Periodic Acid Schiff) allows study of fungal morphology .When a mould is suspected in tissue material isolate ,Fontana Masson stain maybe used as a differential stain .

Typical picture of candida on endoscopy.

Classical pic Aspergillus niger on otoendoscopic view

Aspergillus flavus

Chronic otitis media

Chronic or intermittent purulent otorrhoea through a non-intact tympanic membrane persists for more than 6 weeks despite treatment (*non-intact includes a perforation or a ventilation tube)*

Otitis media is a multifactorial complex disease with 4 stages :

1)Myringitis

2)Acute otitis media

3) Secretory otitis media and

4)chronic otitis media (persistent / intermittent otorrhoea of minimum >6-12 wks duration via perforated TM) (Brooke et al., 2000)

Lot of attention has been given to CSOM not only because of chronicity , morbidity and high incidence but also due to issues of microbial resistance and ototoxicity of OTC topical ear drops .(Haynes ,2002)

CSOM associated with destructive osteitis, granulomations involving middle ear , mastoid ear bowl aremedically not curable and present with foul smelling purulent ear discharge non resolving with usual antibiotics. Character of discharge size of TM middle ear mucosa appearance on otoscopy should confirm diagnosis rather than history of hearing loss and last ear discharge .

All draining ears are not necessarily linked to CSOM. Acute Otitis externa and media also present with discharge and otalgia. Discharge is much less foul smelling, profuse and tends to form mucus threads if tested on cotton gauze. Also, incidence of fever is higher in otitis media cases than otitis externa cases.

CSOM however produces painless ear discharge mucoid in consistency without fever unless accompanied by intracranial infection. But even if ototscopy is not possible , any discharging ear >2- 3 months is likely CSOM, or carries higher risk of resulting in one since both AOM and otitis externa usually may be self limiting .

Overall morbidity and prevalence in developing world ranges <10% of cases (Heyneman, 2000).Topical antibiotic ear drops for achieving a dry ear is the commonest conservative management used for CSOM without cholesteatoma (Loy et al.,2002).

The IMCI guidelines require that children with stiff neck , fever , unable to drink, lethargic , h/o convulsions and emesis be swiftly referred to hospital and given antibiotics as these maybe danger signs for intracranial complications of CSOM.

Cultures of ear discharge isolates are rarely sent for unless failed attempts in response to topical therapy. One of the most widely prescribed drops is Neomycin/polymyxin B/hydrocortisone suspension. In a study in Nigeria , Gram negative isolates had 75% sensitivity to Cefotaxime except for Pseudomonas aeruginosa (Resistance noted)

Diagnosis of fungal infections :

1. Microscopic examination of specimens for fungi is important in diagnosis when possible to obtain before culture . 10% KOH (potassium hydroxide) is most commonly used medium .The slide is left to stand until clear, normally between five and fifteen minutes, in order to dissolve skin cells, hair, and debris.

2. To enhance clearing dimethyl sulfoxide can be added to the slide. To make the fungi easier to see lactophenol cotton blue stain can be added.

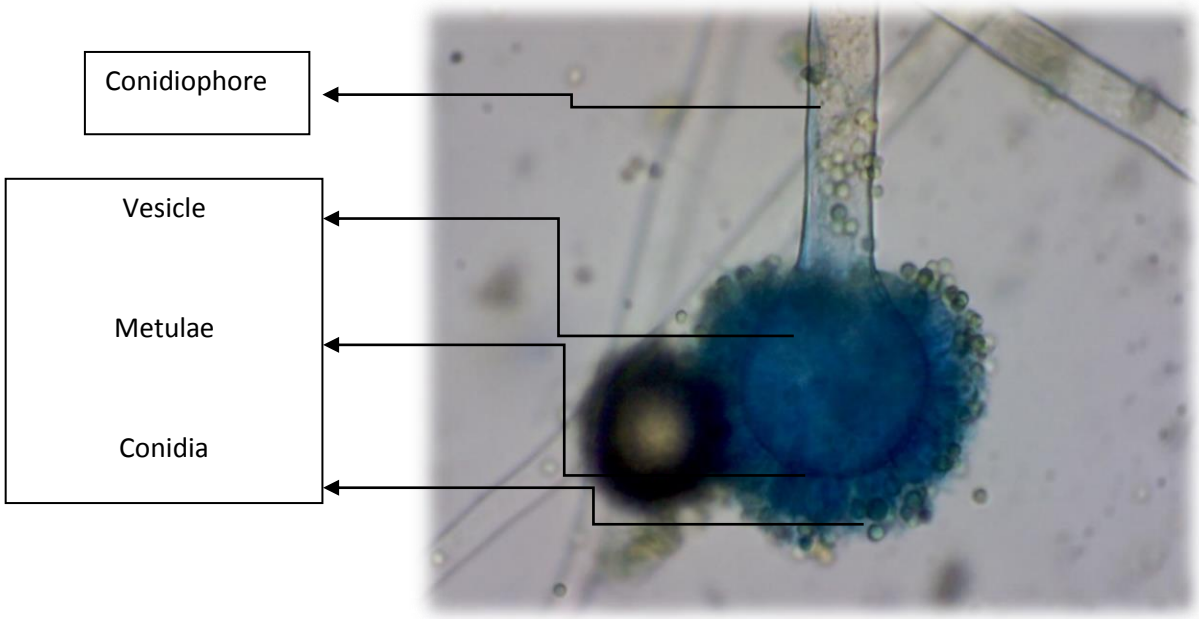
3. The slide is gently heated to speed up the action of the KOH.Adding calcofluor-white stain to the slide will cause the fungi to become fluorescent, making them easier to identify under a fluorescent microscope.

   Different fungal elements like hyphae , pseudohyphae , yeast cells . spores , sclerotic bodies maybe visualised in a KOH wet mount.

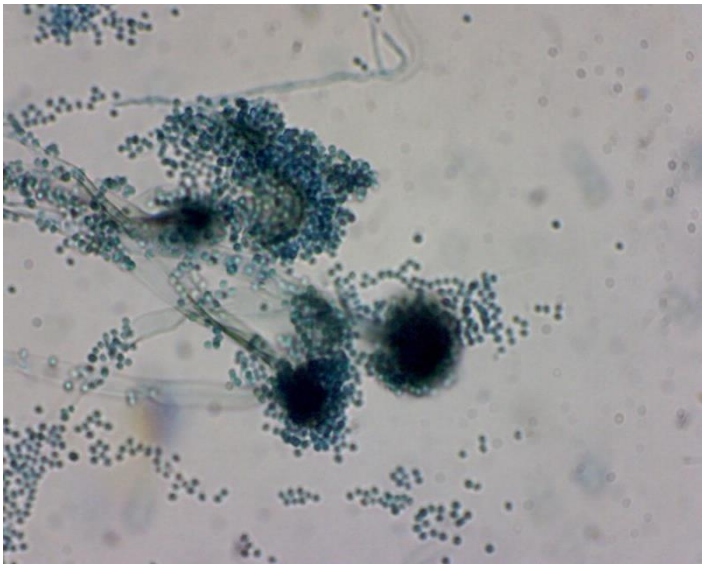
budding yeast cells
Branching pseudohyphae seen under microscope in KOH mount.

Lactophenol cotton blue wet mount : LPCB wet mount preparation is widely and easily followed method for fungal staining and observation .Lactic acid preserves fungal elements while Phenol destroys any live organisms , and cotton blue stains chitin in cell walls of fungi.

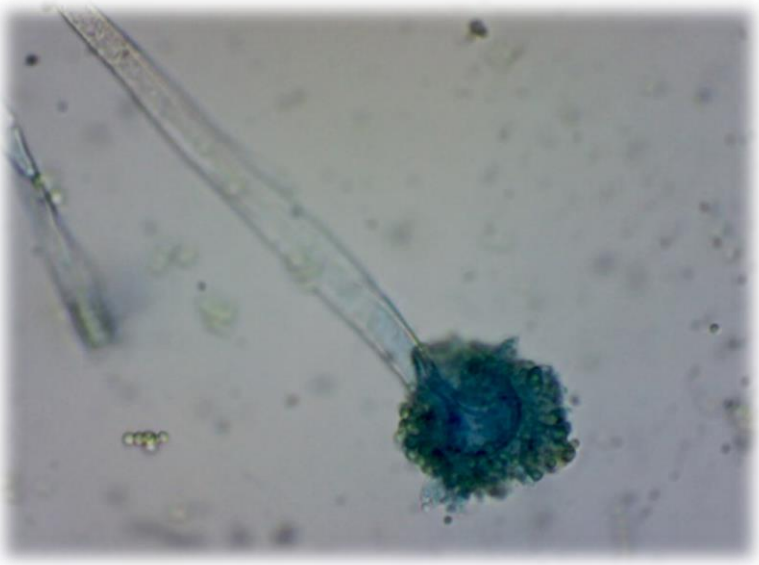
LPCB mount



Vesicle bearing metuale and phalides which forms conidia



Fruiting bodies of Aspergillus with phialides radiating in all directions with black pigmented conidia dispersed .



Aspergillus on LPCB staining showing septate hyphae swollen vesicle giving rise to phialides from which clusters of conidiaa arise

Advantages of Calcofluor over KOH mount

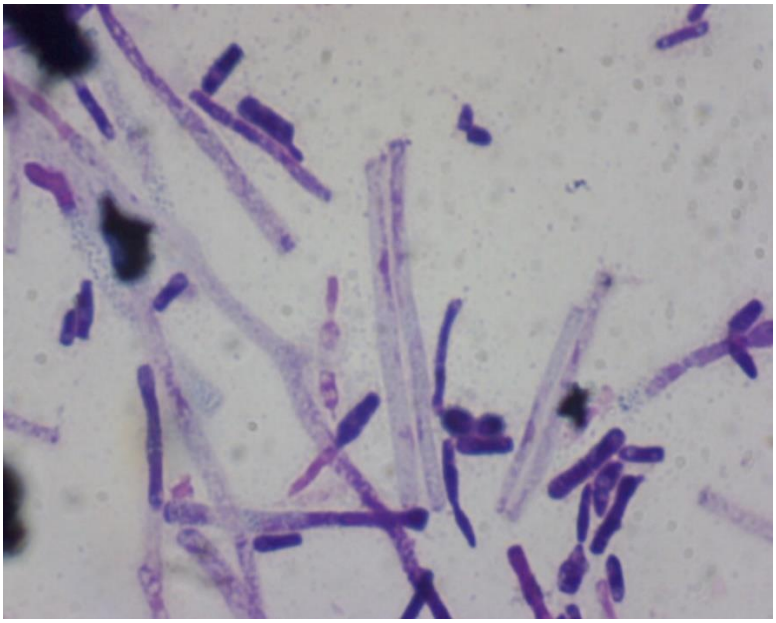
1. Reduced minimum time needed to screen sample.

2. KOH mount requires minimum 3-4 mins for each slide screening.

3. Calcofluor mount can be easily seen for screening in less than 1 minute

4. Reliable, better sensitivity as well as than KOH mount.

5. Helps easy differentiation of background debris .



Gram stain showing individual cells and psedohyphae in Candida .

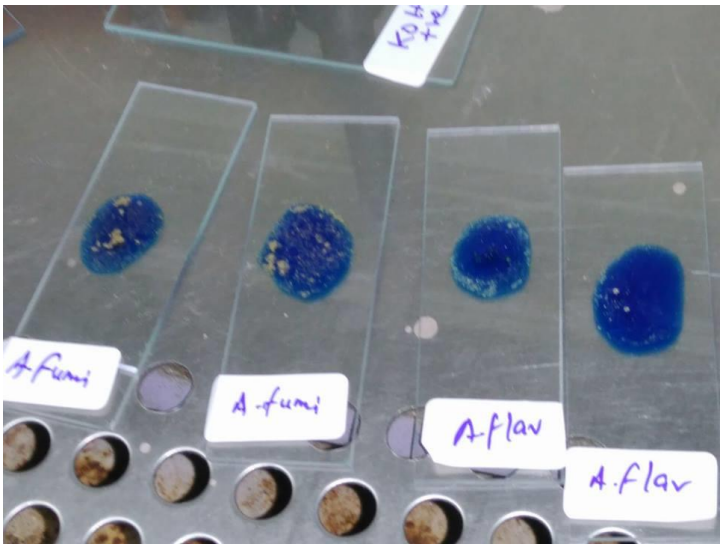
Culture :

Sabouraud’s dextrose agar(SDA) is routinely used for fungal culture.

pH =5.6 does not allow bacterial growth. Chloramphenicol , cyclohexamide and were added as required avoid contamination by bacterial saprophytic fungi super infection. Cultures were subjected to incubation at : 25°C and other at 37 °C to help reveal dimorphism .Cultures underwent incubation for 2-3 weeks, examined for morphology of colonies

(macroscopic)and fungal morphology(microscopically)**.**

Using a sterilized microneedle for sample handling for culture purpose.a drop of LPCB stain applied in clean microscopic slide .drop of 95%ethanol to cover slip after evaporation, the fungal isolate is spread out gently and on the slide over the stain. Examination under microscope after applying cover slip.



AIMS OF STUDY

Despite the fact that climatic condition in Tamil Nadu may encourage fungal infection of middle ear, literature search reveals that not much work has been carried out on various aspects of fungal infection of middle ear. Keeping in view the high prevalence of fungal infection of middle ear in hot, humid and dusty areas, the study was carried out to

- Objectively Isolate, characterize, and identify mycological agents in ear infection of CSOM patients.
- Find out distribution of fungal aetiology in CSOM with and without an intact tympanic membrane.

MATERIALS AND METHODS

100 patients attending ENT OPD and diagnosed to have CSOM were included in the study. Two aural swabs or whenever possible otomycotic debris were taken up on sterile ear swab sticks . From one swab, wet mount preparation in 10% KOH (potassium hydroxide) solution and smear for Grams stain were prepared. to determine the presence or absence of fungal element (hyphae, spores and blastospores).

The second swab / otomycotic debris was directly inoculated into SDA (Sabourad's dextrose agar) medium.

Secretion and pus were collected from 100 patients by two sterile cotton wool swabs taken out from External Auditory Canal or mastoid areas like cholesteatoma. One swab was used for direct microscopy and next for culture examination.. The presence of fungal structures is seen in potassium hydroxide (KOH) wet mounts.

The microscopic examination shows discrete clumps of hyphae with conidiophores. In CSOM caused by A. niger, septate hyphae, sporulating vesicles and abundant black spores are seen. Collected swab was inoculated onto Sabouraud's dextrose agar. The presence of fungal elements in stained smears was confirmed by growth of

fungal colonies.Yeast like organisms like Candida may grow partly as chains of budding elongated cells joint at end (pseudomycelium and pseudohyphae).

KOH MOUNT: KOH dissolves keratin and cellular material while not affecting fungi.Specimen is placed on a slide , a drop of 10-20% KOH is added, covered with a coverslip , left for 20 min in incubator at 37 °C to digest keratin , then examined microscopically .

SDA culture :qualitative method of testing for most fungi. It is a general purpose peptone medium with dextrose (supporting growth of the fungi)and antibiotic like Chloramphenicol and cycloheximide prepared at a pH of 5.6 favouring growth of many species mainly dermatophytes. Agar should optimum moisture for inoculating specimen with sterile loop kept for observation in 25-30°C .

Cultures are examined atleast weekly and should be observed till min. 4 weeks before reporting negative result.

RESULTS

In this study , analysis of 100 patients with CSOM was done in ENT dept of PSG Institute of Medical Sciences &Research over a period of 2 years to determine incidence of otomycosis in patients with CSOM .

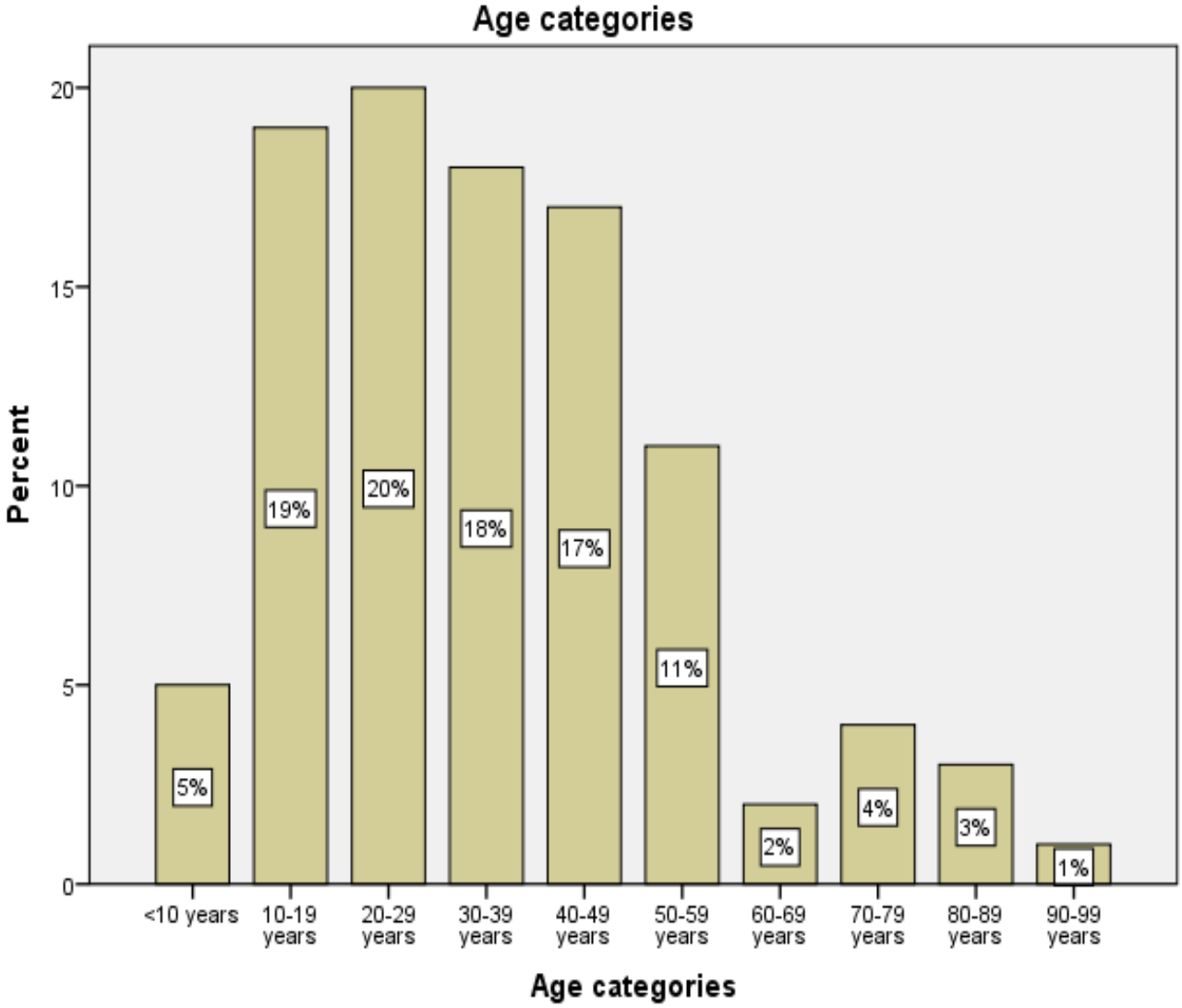
Clinical data was collected by means of a proforma and the observations made were analysed with a Master Chart as given in Annerxure.

The results have been evaluated primarily keeping in mind the aims of the study using Microsoft Excel and SPSS softwares.

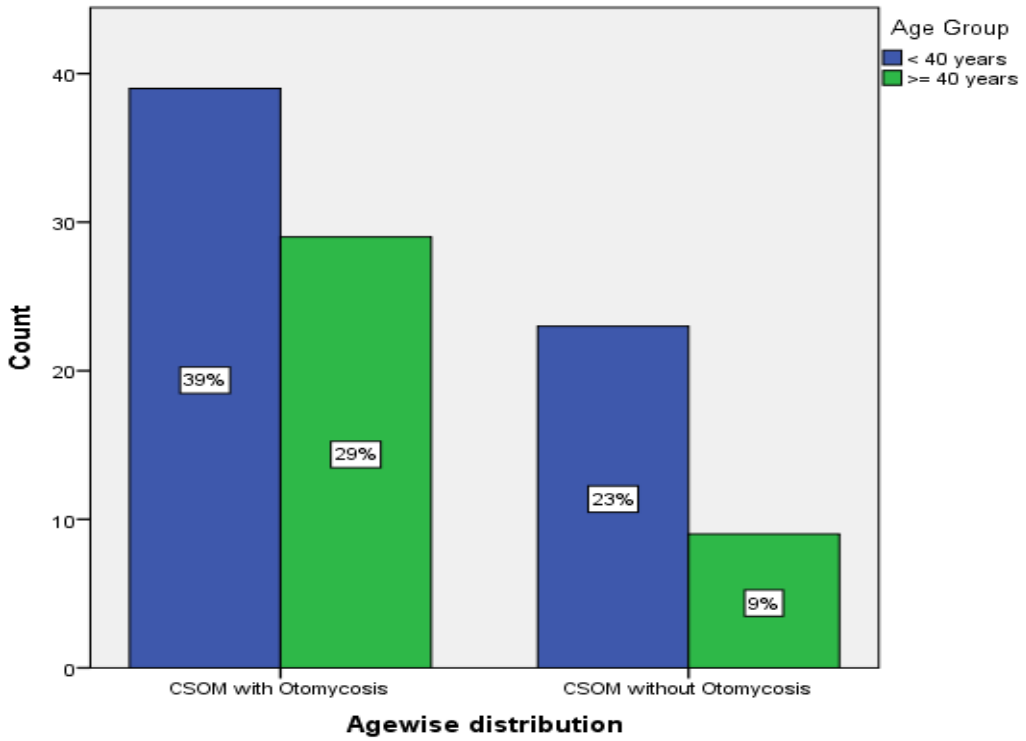
In our study 70% of patients evaluated for CSOM were found to have coexisting otomycosis.

	Frequency	**Percent**
<10 years	**5**	**5.0**
10-19 years	**19**	**19.0**
20-29 years	**20**	**20.0**
30-39 years	**18**	**18.0**
40-49 years	**17**	**17.0**

50-59 years	11	11.0
60-69 years	2	2.0
70-79 years	4	4.0
80-89 years	3	3.0
90-99 years	1	1.0
Total	100	100.0



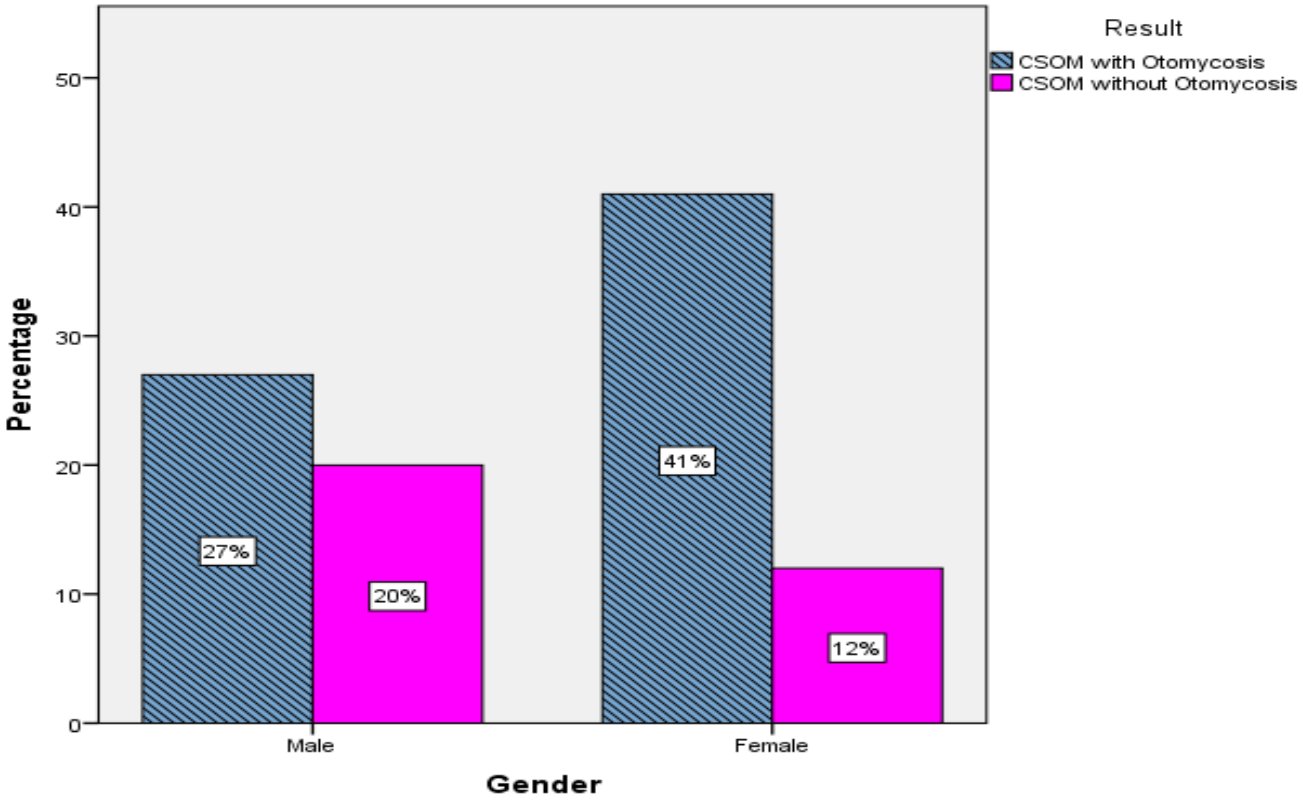
Age-wise distribution	CSOM with Otomycosis	CSOM without Otomycosis	p value
< 40 years	39 (62.9%)	23 (37.1%)	0.24
≥ 40 years	29 (76.3%)	9 (23.7%)	



Overall age distribution in our study

From the table above it is evident that maximum incidence of otomycosis is in age group 20-29 yrs (20%) followed closely by 19% (10-19 yrs age group) 18%(30-39 yrs) with the least incidence being post 60 yrs of age.Overall incidence of CSOM

coexisting with otomycosis is 39% before age of 40 years which is high when compared to 23% cases with CSOM only. In age group more than 40 years, coexisting otomycosis and CSOM is present only in 7% cases while CSOM alone exists in 23%.



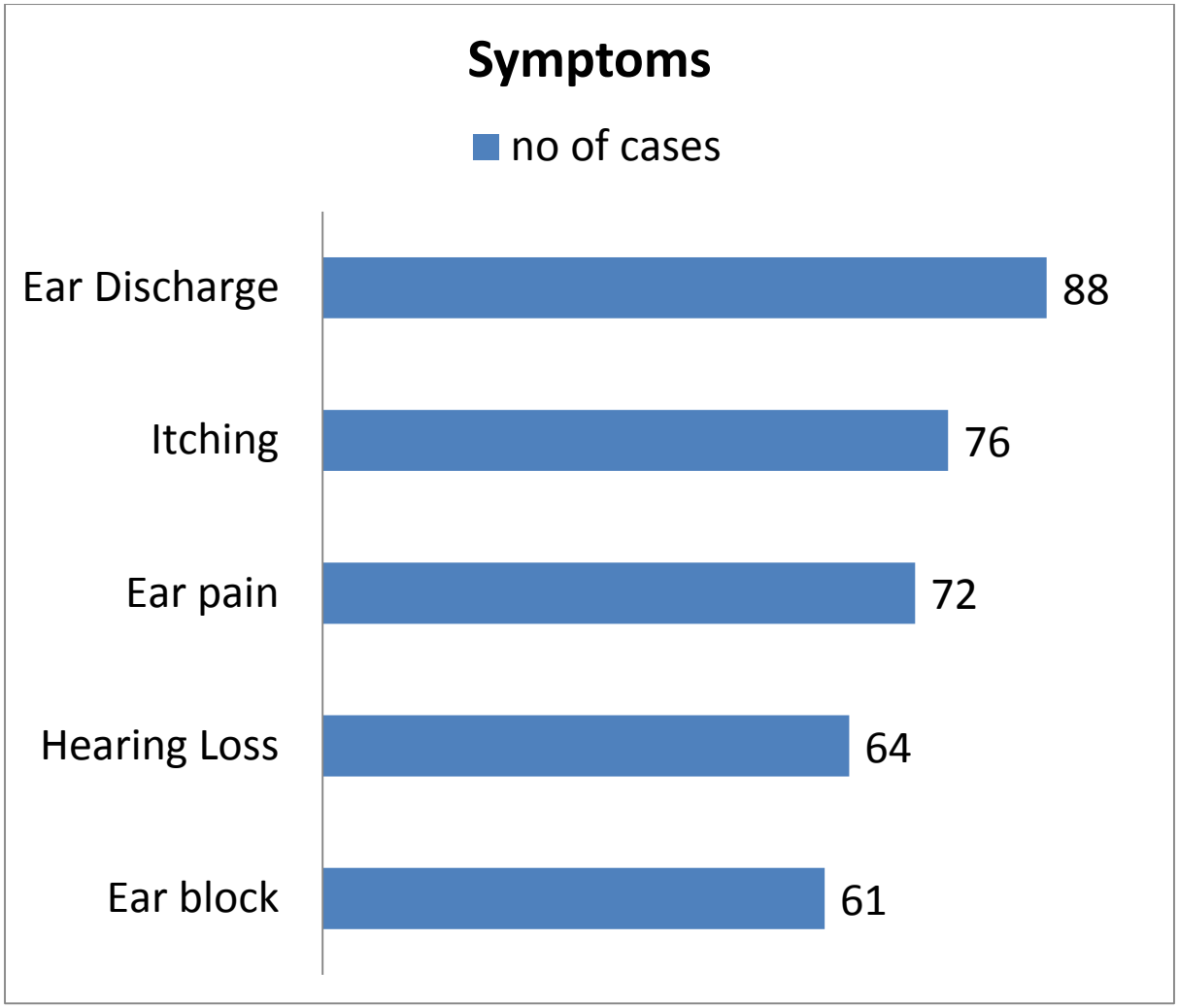
In our study females have higher incidence of coexisting otomycosis with CSOM 41% compared to 27% in males .whereas incidence of CSOM alone is much higher in males (20%) compared to 12% in females. P value is significant <0.05

Gender	**CSOM with Otomycosis**	**CSOM without Otomycosis**	**p value**
Male	**27 (57.4%)**	**20 (42.6%)**	**0.033***
Female	**41 (77.4%)**	**12 (22.6%)**	

Clinical features of CSOM with Otomycosis

Symptoms	Pre operative incidence
Ear Discharge	88
Hearing Loss	64
Itching	76
Ear pain	72
Ear block	61

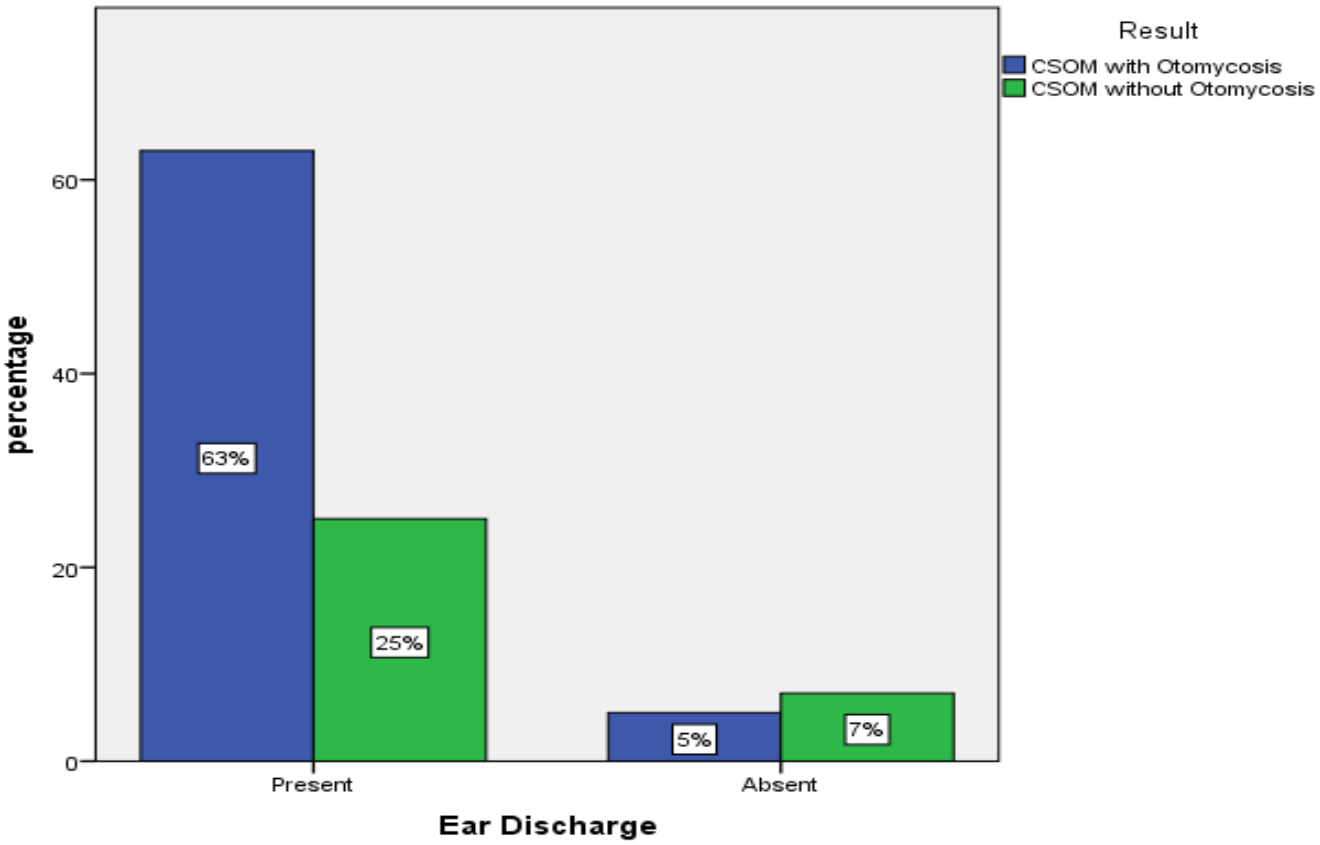
Most common complaint in our study at time of presentation was ear discharge (88%) followed by itching (76%) and ear pain (72%). The other complaints included hearing loss and ear block 64% and 61% respectively.



Overall distribution of symptoms in our study population

Ear Discharge	CSOM with Otomycosis	CSOM without Otomycosis	Total		p value
Present (n=88)	63 (71.6%)	25 (28.4%)	88		0.037*
Absent (n=12)	5 (41.7%)	7 (58.3%)	12		
Total	68	32	100		

Odds ratio = 3.53 with 95% C.I =(1.02 – 12.16)

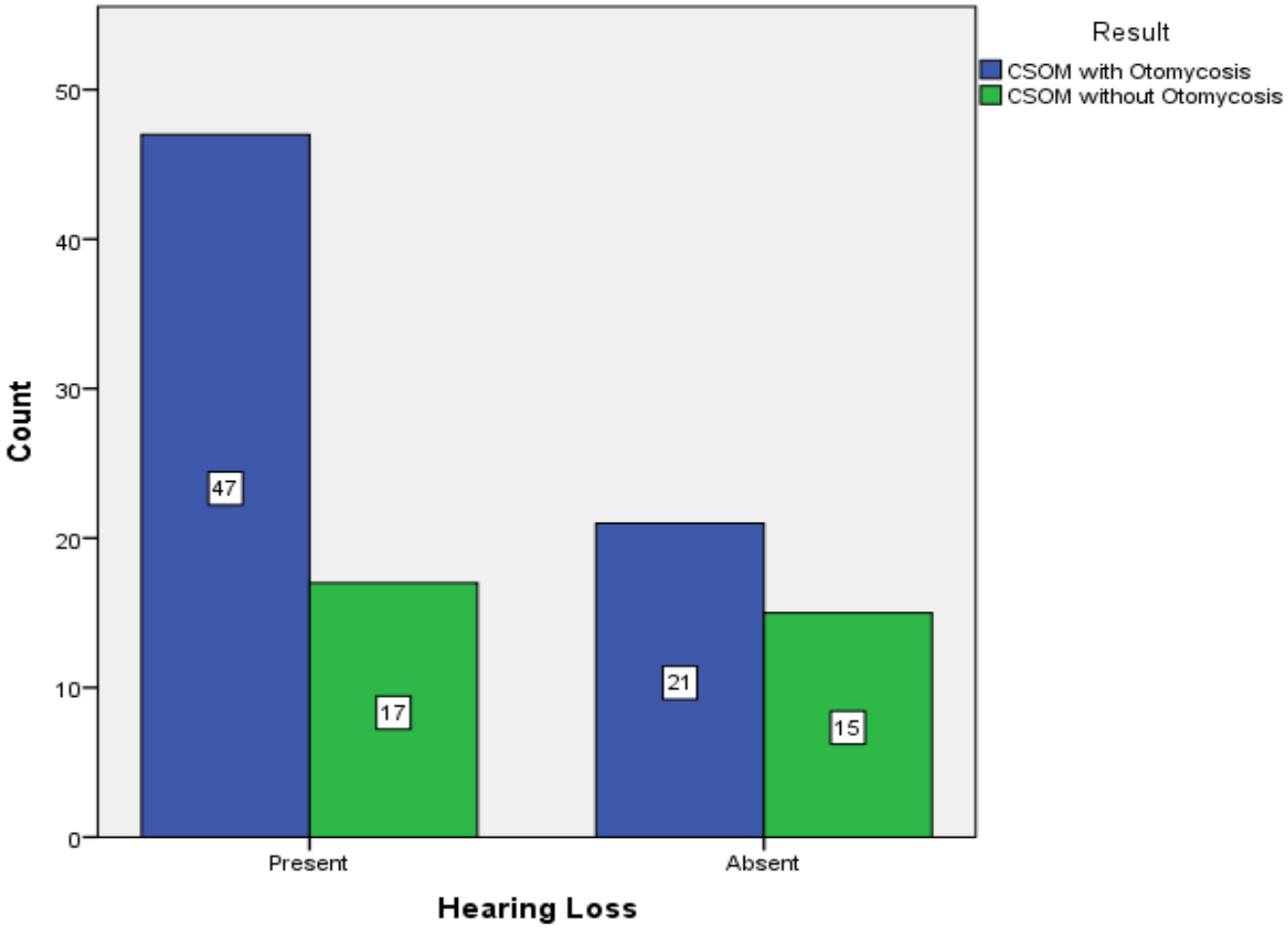


The chance of a patient presenting with ear discharge having otomycosis as diagnosis is 3.5 times than not having otomycosis and is statistically significant.

Hence ear discharge as a significant symptom of otomycosis is statiscally significant(p value <.05)

Hearing Loss	CSOM with Otomycosis	CSOM without Otomycosis	Total	p value
Present (n=64)	47 (73.4%)	17 (26.6%)	64	0.12
Absent (n=36)	21 (58.3%)	15 (41.7%)	36	
Total	68	32	100	

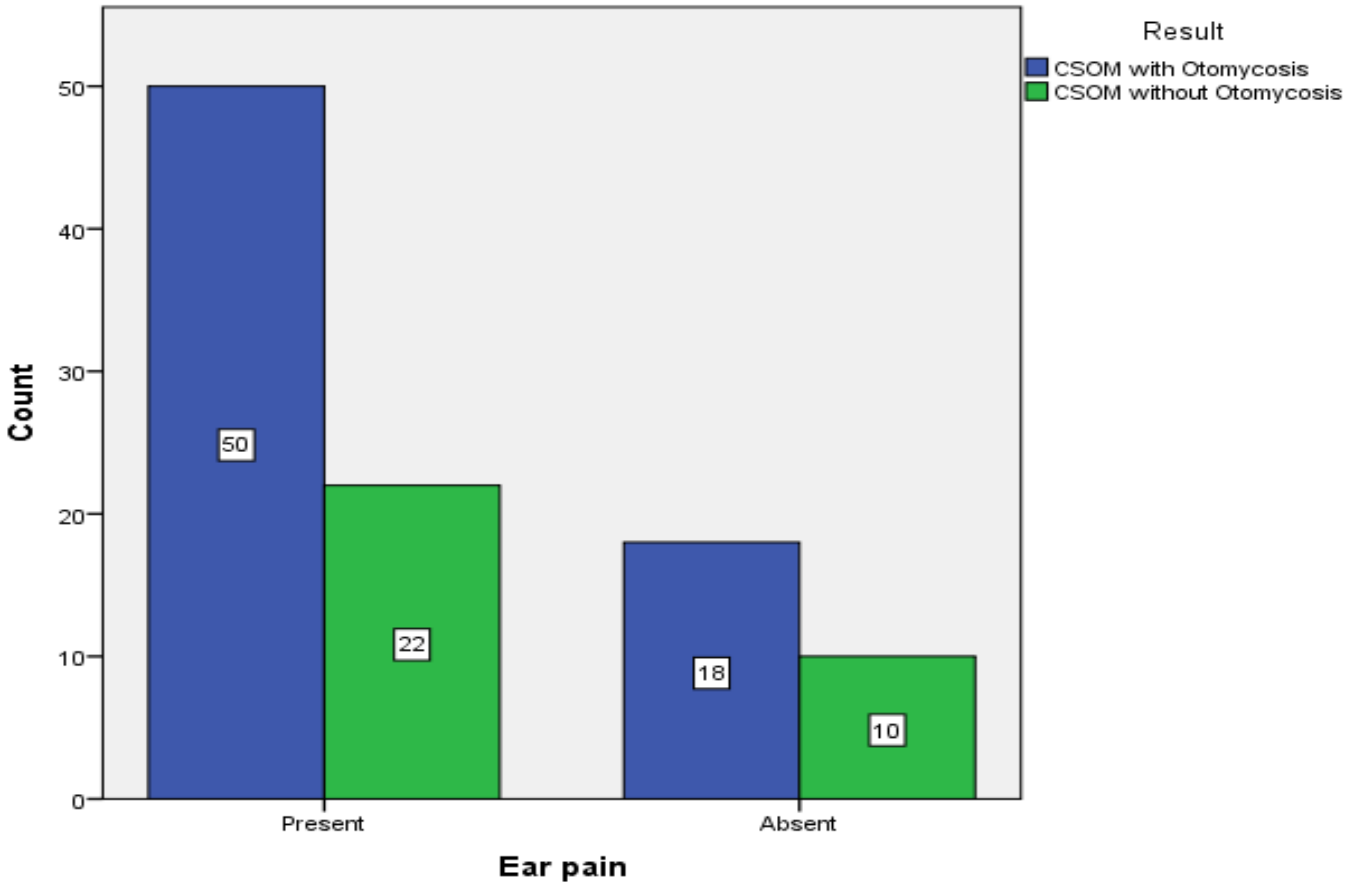
Hearing loss as a symptom for otomycosis is not as statiscally relevant when compared with CSOM cases alone (p value >0.05)



Ear pain	CSOM with Otomycosis	CSOM without Otomycosis	Total	p value
Present (n=72)	50 (69.4%)	22 (30.6%)	72	0.620
Absent (n=28)	18 (64.3%)	10 (35.7%)	28	
Total	68	32	100	

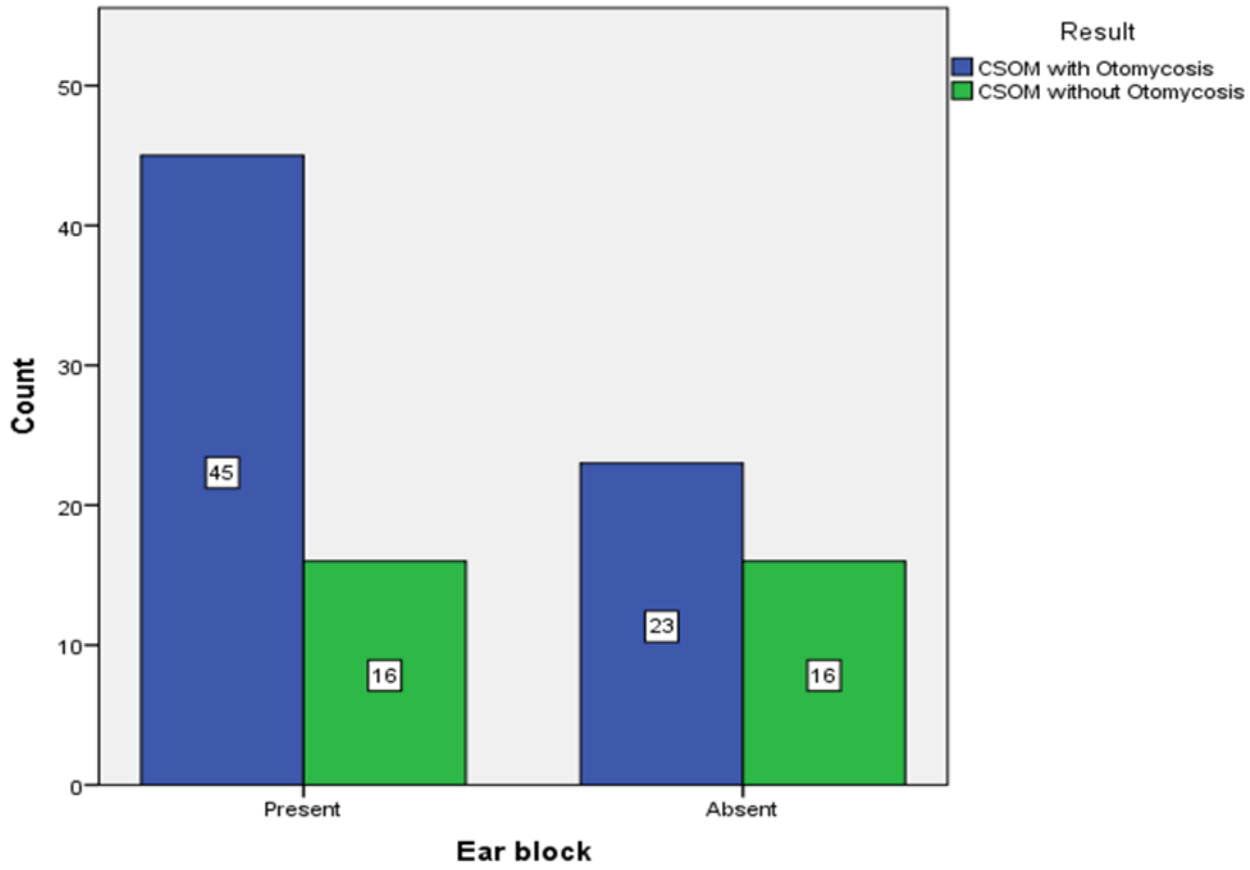
Odds ratio =1.26

The chance of a patient presenting with Ear pain having otomycosis as diagnosis is 1.3 times than not having otomycosis and is not statistically significant.



Ear pain does not show as a major significant symptom for otomycosis with SOM cases when compared to CSOM cases alone in this study .

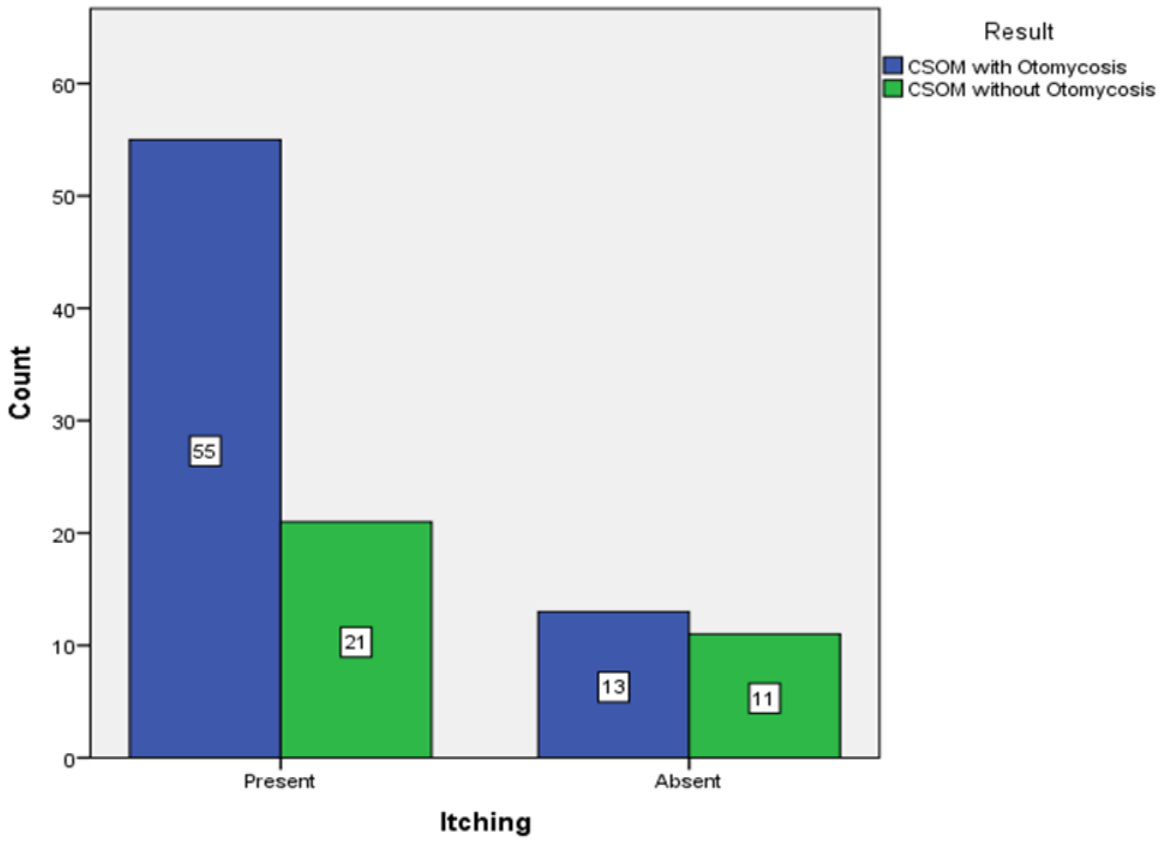
(p value >0.05)



Ear block	CSOM with Otomycosis	CSOM without Otomycosis	Total	p value
Present (n=61)	45 (73.8%)	16 (26.2%)	61	0.122
Absent (n=39)	23 (59%)	16 (41%)	39	
Total	68	32	100	

Odds ratio =1.96 .The chance of a patient presenting with Ear block having otomycosis as diagnosis is 1.96 times than not having otomycosis and is not statistically significant.

Itching	CSOM with Otomycosis	CSOM without Otomycosis	Total	p value
Present (n=76)	55 (72.4%)	21 (27.6%)	76	0.096
Absent (n=24)	13 (54.2%)	11 (45.8%)	24	
Total	68	32	100	



Odds ratio =2.22

The chance of a patient presenting with Itching having otomycosis as diagnosis is 2.2 times than not having otomycosis and is not statistically so significant.

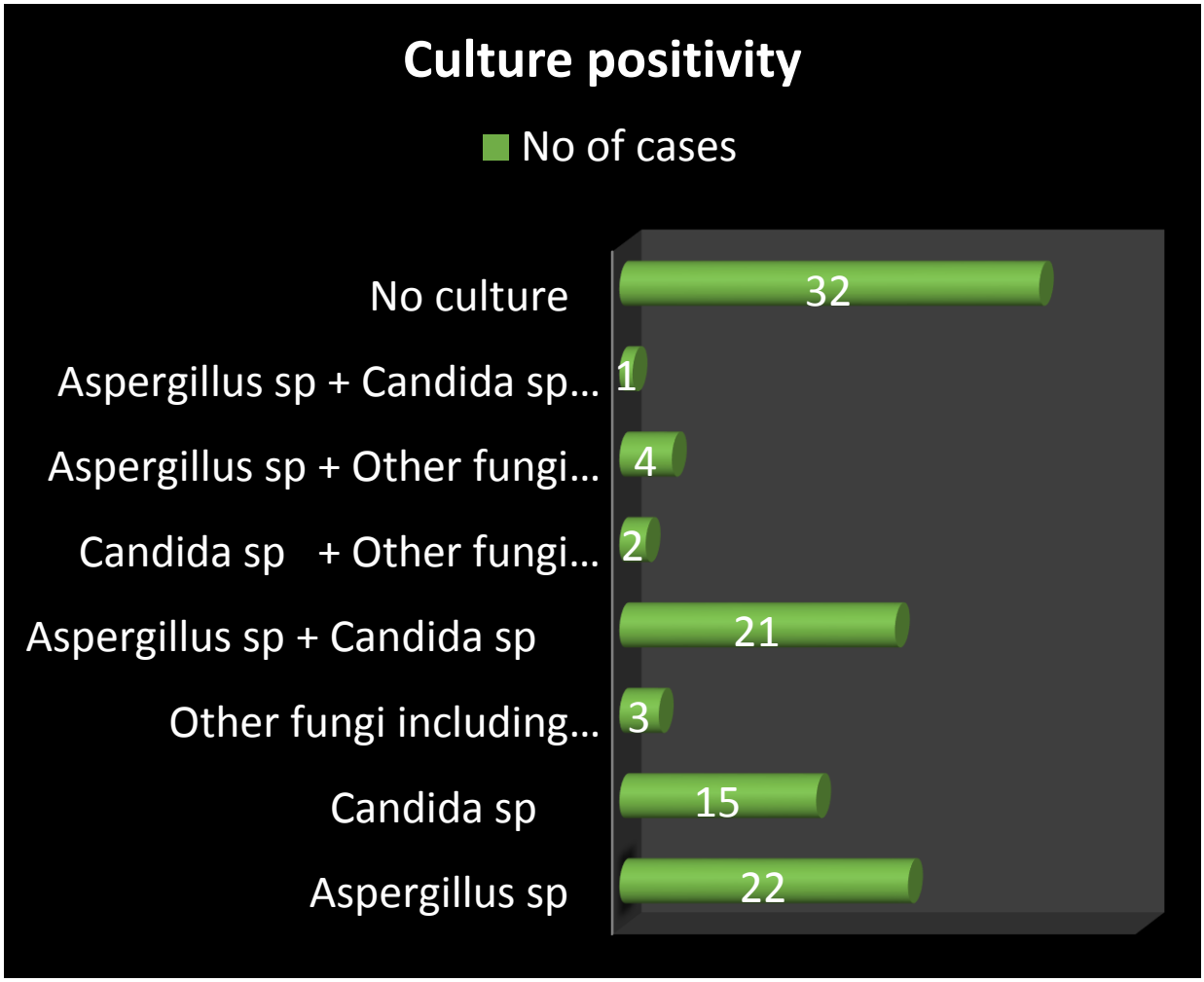
Fungal culture shows :

Fungus	No.of Cases	Percentage(%)
Aspergillus sp	22	22
Candida sp	15	15
Other fungi including dermatophytes	3	3
Aspergillus sp + Candida sp	21	21
Candida sp + Other fungi including dermatophytes	2	2
Aspergillus sp + Other fungi including dermatophytes	4	4
Aspergillus sp + Candida sp + Other fungi including dermatophytes	1	1
No culture	32	32
Total	100	100

Aspergillus as the most commonly identified fungus 22% .followed by aspergillus with Candida species .21%

Candida as a fungus alone was foundin as high as 15% of cases.

A mixed growth of Aspergillus with other species such as dermatophytes were also significant 4%. While other combinations like Candida with dermatophytes (2%) and lastly Aspergillus , Candida and other fungal growth is observed in 1% cases.Culture report failed to show fungal growth in 32% cases.



The number of fungal positive cases in our study was 68%.

KOH smear	CSOM with Otomycosis	CSOM without Otomycosis	p value
Present (n=81)	61 (75.3%)	20 (24.7%)	0.001*
Absent (n=19)	7 (36.8%)	12 (63.2%)	

KOH smear/ microbiological finding

Odds ratio = 5.23 with 95% C.I =(1.81 – 15.09)

The chance of a patient having KOH smear positive to have otomycosis as diagnosis is 5.2 times than not having otomycosis and is statistically significant

DISCUSSION

Review of literature shows otitis media to be a common medical problem in India [18,19]. Controversies in the past regarding existence of fungal cause of CSOM existed , now it has been proved clinically positive entity [8] possibly because of manifold increase in incidence and the variety of pathogenic fungi in recent years. [9] Morbidity is affected as diabetes, , malignancy, , chemotherapy, HIV infection, irresponsible over the counter antibiotic use (topical or systemic) and steroid administration renders the individual prone to fungal infections of external as well as middle ear [10]

Age:

Relatively less number of available studies regarding fungal CSOM. In ourstudy, highest incidence was recorded in 2nd , 3rd and 4th decades of life 57% of the study population and this observation bears similarity to other studies by various authors Mohanty JC et al[9], Chander J et al [10] Paulose KO [11] and Kaur R[12.Only 10% patients had otomycosis post age of 60 yrs while only one patient in age group 90-99yrs had otomycosis .[15]

The incidence decreased with increase in age post 45-50 yrs of age. Higher incidence in young adults may be attributed to more exposure to airborne fungal spores in daily work and lifestyle while extreme age groups are relatively spared.

Gender : In present study, females 41(77.4%) had otomycosis compared to 27(57.4%) .In CSOM without otomycosis, males suffer more 20(42.6%) compard to females 12(22.6%). In other studies , males predominantly suffer more than females as far as fungal infection in CSOM is concerned .Observation of various authors Laxmipati , Baskaran 55.80%, EO Nwankwo, AD salisu 57.5%, Rachna et al 55.55%, Gulati et al 67.50% shows more male affected with otomycosis.

In contrast, the findings of present study correlate well with the observation of Reena Roy et al[11] who found females 57% were most commonly affected. In one “Mycological study of otomycosis” by Rajeshwari Raoet al. in Karnataka,among94 specimens of fungal isolates, males were41(43.6%) whereas 53 (56.4%) were females. Females are more affected than males in our case probably because they are more exposed to hot , humid climates in household , lifestyle and habit of self probing with unsterile sticks , cotton

buds , putting various oils(coconut oils) in EAC and less tendency to follow up in OPD for minor itching and ear block symptoms.

Reports of “sporostatic” action of coconut oil may actually preserve the viability of fungal elements present in EAC, indirectly contributingto the problems.

Most common Symptoms

In our present study , ear discharge and itching (pruritis) were primary complaints on 1st OPD visit (88%) and 76% respectively. Which correlate well with studies done in North Karnataka by Archana et al in a study on clino mycological profile of Otomycosis .Hearing loss and aural block are less commonly found in present study which is similar to study by Archan S ,Channabasawaraj et al.,which shows aural block ,hearing loss and tinnitus in lesser number .72% of otomycosis cases had itching while only 21% withonly CSOM complained of the same.

Most of our patients had unilateral disease as similar to other studies.

Present report shows 3.5 times higher incidence of otorhoea , 63% in otomycosis with CSOM than 17% in cases with only CSOM as in

other studies upto 90% being primary complaint [8,75]47% had hearing loss as a primary complaint in our study which is comparable to study done in Hyderabad (Chapparbandi, Farha Naaz) as 35% had deafness.

Present report has 45% ear block and deafness 47% comparable to studies published in Pakistan Journal of Medical Sciences by Khurshid Anwar etal. Which shows Ear block 30.8% , hard of hearing 50%.

High incidence of *A. niger* in external ear is common finding in most studies.Mostly aspergilli are not affected by wax (cerumen)[76].First described by Cramer(in 1859),A. niger was isolated from human ear by him[77,78], *Aspergillus*was the primary cause of otomycosis [78,79].Aspergillus in our study has accounted for 48% of all cases positive for fungus.(22% Aspergillus only , 21% with Candida spp, 5% with other species .

Candida in our case series was detected in 41% cases (15%only Candida ,

21% mixed with Aspergillus and only 3% with other saprophytic fungi) which is comparable to study In Brazil showing 51% . In one study in Tropical Medicine journal in Africa by Deepak Juyal et al.,

Aspergillus species accounted for 47% of the cases, while 41% of the tests shows Candida species.

This report aims to highlight the most common fungal etiology for otomycosis in our setting. Aspergillus spp(niger more than flavus and fumigatus) with all cases responding effectively to agents like clotrimazole , neomycin post proper aural toileting and following precautions to keep a dry ear . otomycosis is a increasingly common problem in Tamil Nadu ,Hyderabad Karnataka region .

Prevalence of increase in fungal infections during and humid conditions has been reported in multiple studies [12],[13],14]

We suggest a definite search for fungi is desirable in all cases of CSOM.

The diagnosis based on microbiological findings should be prompt.

The present study was undertaken to know the incidence of fungal infection in chronic suppurative otitis media, to know the pattern of fungal flora in chronic suppurative otitis media.There is emerging need to follow up patients with more case studies of the same. In cases of CSOM, not responding to local antibiotics drops superimposed fungal infection must be suspected. There is a great disproportion of otomycosis coexisting with CSOM cases and the number of studies regarding clinical profile of the same .

Otitis media is described as inflammation of mucoperiosteal lining of middle ear, with or without intact tympanic membrane.It is one of most common childhood infections and a leading reason for antibiotic prescriptions in ENT .

Otomycosis (fungal otitis externa) amounts for significant number of OPD cases as well .The disesase is highly prevalent in South Asian region. [20]Chronic otitis media and CSOM are few of the common conditions encountered in a general otolaryngology clinic setting and its prevalence has been quoted to range from 9% to27.2% [2, 6] among patients who present with signs and symptoms of otitis externa and up to 30% [18,19] in patients with discharging ears. Distribution worldwide with more prevalence in the humid, and dusty areas of the tropics and [2,6,18,19].Fungi can either be the primary pathogen or be superimposed on bacterial infections or can be secondary pathogen in previously perforated tympanic membrane. It is mainly characterized by pruritus, otalgia, aural fullness, hearing impairment and tinnitus. Various predisposing factors have been proposed for fungal ear infection, including immunocompromised host, steroid usage, trauma, swimming, ear picking, use of headwear, use of oils, instrumentation of ear, fungal infection elsewhere in the body like dermatomycosis and

malnutrition in children[1-7] . Wide spectrum of fungal agents such as Aspergillus, Penicillium, Mucor, Rhizopus, Scopulariopsis, Absidia and Candida are involved, species of Aspergillus and Candida being the most common .*Aspergillus* and *Candida* spp are the most commonly isolated fungi in India in most studies .

Usual presentation of CSOM with otomycosis is similar to symptoms of acute otitis externa in some cases . The diagnosis of otitis externa relies on the patient's history, otoscopic examination under microscopic control, imaging studies .History of ear probing (accidental EAC injury) , immune deficiency status are not always present in many patients who initially complain mainly of minor ear itching. The widespread indiscriminate haphazard use of antibiotics and poor follow up among patients may result in persistence of coexisting low grade infection even after initial symptoms subside.

. In general, Fungi causing otomycosis vary geographically from yeasts, moulds to dermatophytes , Occasionally, other genera involved are Absidia, Fusarium, Gymnoascus, Exophiala,Mucor, Scopulariopsis, [62-68]Aspergillus, in some publications has shown to be most frequently involved [69] while others Candida is predominant[70]. Almost similar findings are recorded in Indian subcontinent [82-85].

Few case series however do report incidence of Candida has been reported to be higher than Aspergillus [86,87].High incidence of otomycosis in Indian subcontinent has been reported in past studies .Delay in proper diagnosis and severity of the disease prolong recovery .There is usually good recovery in immunocompetent patients on institution of topical antifungals. High clinical suspicion initially can prevent development of risk of recurrence and reduce unnecessary usage of antibiotic and steroids for extended periods .Primary and secondary otomycosis clinical criteria can be standardized for better reporting of cases.

Approx. 5-25% of otitis externa may progress to otomycosis. And 10% of ases of external otitis are due to otomycosis.

Management :

Aspergillus and *Candida* spp are the most frequently isolated fungi in patients with otomycosis. Topical antifungals, such as clotrimazole, miconazole, bifonazole, ciclopiroxolamine, and tolnaftate, are potentially safe choices for the treatment of otomycosis, especially in patients with a perforated eardrum.

The oral triazole drugs, itraconazole, voriconazole, and posaconazole are effective against *Candida* and *Aspergillus*, with good penetration of bone and the central nervous system.

Recommendation in refractory otomycosis due to its mostly lack of ototoxicity antifungal preparation Bifonazole was in common use during 80s[24,25]. bifonazole 1% solution has similar potency in comparison to miconazole or clotrimazole . It has retarded fungal growth in most cases with efficacy of up to 100% [29,30].

Boric acid maybe used to treat fungal and yeast infections caused by Candida sp. Tolnaftate 1% solution can also be easily instilled into the ear [26]

5-Fluorocytocine (flucytosine) penetrates fungal cells ,fluorouracil is formed as active drug, which competes with uracil and hence fungal RNA formation [27,28]. In our setting, all patients were given topical Clotrimazole , Neomycin with steroid combination and followed up weekly for atleast 4 wks or when patient did not turn up for review whichever was later.In follow up re-examination was done for alleviation of symptoms and most of them showed positive response.

Bassiouny et al. had reviewed the efficacy of topical antifungal agents to conclude that Clotrimazole and econazole were mostly effective

Systemic antifungal therapy:

In patients who fail response to topical therapy have invasive external otitis , systemic antifungal measures maybe used. Itraconazole may be tried in superficial external otitis but in case of invasion, which includes TM perforation voriconazole maybe useful. If perforation of TM does not heal easily , patients may need Tympanoplasty (Wang et al, 2005).

Resistance to Itraconazole has been seen in otomycotic isolates of Aspergillus fumigatus and Aspergillus niger (Kaya et al, 2007, Snelders et al, 2008).

One study shows topical ketoconazole effecting better resolution of symptoms with lower recurrence, whereas another study shows voriconazole to be more potent than itraconazole in vitro against *Aspergillus* spp. *Candida albicans* .

Other authors suggested that in all cases of CSOM , mere instillation of topical ear drops (antibiotic/steroid) is not mandatory .It may lead to bacterial flora suppression and flaring of

normal commensal flora of EAC causing superinfection .Otologists need to develop a sense of suspicion in refractory ear discharge cases not responding to conventional treatment .At the very onset , aural toileting is justifiable to reduce the prevalence of fungi in endemic regions.

Complications :

Otomycosis is frequently seen more in patients with immunocompromise with more chances of complication and recurrence. They also require longer duration of treatment and complications.

Infrequent but reported complications include

1. ear drum perforation ,
2. Hearing loss (not fully reversible)
3. Invasive temporal bone infection
4. Meningoencephalitis
5. Fungal otomastoiditis

Malignant otitis externa has potential to rarely present as aggressive angioinvasive temporal bone infection.(otomastoiditis in case of invasive Aspergillus spp. More commonly nowadays found in

patients of immunodeficiency. In these situations , recommended treatment is surgical debridement with systemic antifungal (Amphotericin B) for 3-4 wks

Tympanic membrane involvement is likely a consequence of fungal inoculation in the most medial aspect of the external canal or direct extension of the disease from adjacent skin

Otologist should remain alert for otomycosis and should consider obtaining hematological investigations and fungal cultures when this disease is suspected in immunocompromised host.

LIMITATIONS OF STUDY

1. Large number of patients eligible for study were lost to follow up and a few participants had to be excluded as swabs for culture could not be taken at time of initial presentation due to treatment previously with topical ear drops and lack of visible discharge in ear .
2. There was no separate culture media used for transportation of swabs to microbiology lab .
3. Most of the cases were chronic, so long term changes in predominant fungal flora could not be taken into account in all cases.
4. It was not possible to get confirmatory culture results in a few

   due to inability to clean sample from ear discharge (eg in cases contaminated in excess with wax).

CONCLUSION

A. Incidence of otomycosis superimposed on CSOM in our study is 68% of all cases tested .

B. Incidence was higher in middle age and young adults than both extremes of age groups.

C. Ear discharge , itching in EAC ,ear pain were the most significant complaints in all cases

D. Aspergillus is the primary fungi species effecting otomycosis in our study closely followed by Aspergillus spp with Candida, and subsequently major cause being Candida.

E. KOH mount is effective as a primary means of screening for fungi giving in present context sensitivity of 89.7% . but specificity for true negative cases was low 37.5%

BIBLIOGRAPHY

1. 1. Chronic suppurative otitis media : burden of illness and management options. I.Acuin, Jose II.World Health Organization
2. Z.B.V.D.S.Pontes,A.D.F.Silva,E.D.O. Lima. Otomycosis: a retrospective study. Brazilian Journal of Otorhinolaryngology. 2009;75(3):367–370.
3. Prasad SC , Kotigadde S, Shekhar M,Thada ND, Prabhu P, D' Souza T, Prasad KC. Primary Otomycosis i n the Indian Subcontinent: Predisposing Factors, Microbiology, and Classification. Hindawi Publishing Corporation,International Journal of Microbiology,Volume 2014, Article ID 636493.
4. Gokale SK ,Suligavi SS, Baragundi M, Anushka D4, Manjula R. Otomycosis : A Clinico Mycological Study. Int J Med Health Sci. 2013; 2(2): 218-23.
5. 3. R. Munguia and S. J. Daniel, "Ototopical antifungals and otomycosis: a review. International Journal of Pediatric Otorhinolaryngology/ 2008 ;72(4): 453–9. 4. Pradhan B, Tuladhar NR, ManAmatya R. Prevalence of otomycosis in

outpatient department of otolaryngology in Tribhuvan University Teaching Hospital, Kathmandu,Nepal. Annals of Otology, Rhinology and Laryngology.2003; 112(4): 384–7

6. Viswanatha. B et al. Otomycosis in immunocompetent and immune compromised patients; comparative study and literature review, ENT Journal 2012 Mar; 91(3):114-21

7. Paulose KO, Al Khalifa S, Shenoy P, Sharma RK. Mycotic infectionof the ear (otomycosis) : a prospective study. J Laryngol otol, 1989;103: 30-5.

8. Pradhan B, Tuladhar NR, Amatya RM. Prevalence of otomycosis in outpatient department of otolaryngology in Tribhuvan University Teaching Hospital, Kathmandu, Nepal. Ann Otol Rhinol Laryngol 2003;112:384-7.

9. Viswanatha B, Naseeruddin K. Fungal infections of the ear in immunocompromised host: A review. Mediterr J Hematol Infect Dis 2011;3:e2011003

10. Talwar P, Chakrabarti A, Kaur P, Pahwa RK, Mittal A, Mehra YN. Fungal infections of ear with special reference to chronic suppurative otitis media. Mycopathologia 1988;104:47-50.

11. Pontes ZB, Silva AD, Lima Ede O, Guerra Mde H, Oliveira NM, Carvalho Mde F, *et al*. Otomycosis: A retrospective study. Braz J Otorhinolaryngol 2009;75:367-70.

12. Fasunla J, Ibekwe T, Onakoya P. Otomycosis in western Nigeria. Mycoses 2008;51:67-70

13. Kumar A. Fungal spectrum in otomycosis patients. JK Sci 2005;7:152-5.

14. Ozcan KM, Ozcan M, Karaarslan A, Karaarslan F. Otomycosis in Turkey: Predisposing factors, aetiology and therapy. J Laryngol Otol 2003;117:39-42.

15. Healy GB. Otitis media and middle ear effusions. In: Ballenger JJ. The Ear, Williams and Williams. Baltimore; 1996: 1003-9.

16. Seely DR, Gloyd SS, Wright ADO, Susan JN. Hearing loss prevalence and risk factors among Sierra Leonean Children. Arch Otolaryngol Head Neck Surg 1995;121:853-58.

17. Bluestone CB, Klein JO. Epidemiology, Otitis media in infants and children.. WB Saunders, Philadelphia; 2001: 58-78

18. Pradhan B, Tuladhar NR, ManAmatya R. Prevalence of otomycosis in outpatient department of otolaryngology in Tribhuvan University Teaching Hospital, Kathmandu,Nepal. Annals of Otology, Rhinology and Laryngology.2003; 112(4): 384–7.

19. B.Barati, S.A.R. Okhovvat, A. Goljanian, M.R.Omrani. otomycosis in central iran: a clinical and mycological study. Iran Red Crescent Med J. 2011; 13(12):873-6.

20. Parveen S, Rao R. Aerobic bacteriology of Chronic Suppurative Otitis Media (CSOM) in a teaching hospital. J Microbiol Biotechnol Res 2012;2:586-9.

21. Kumar H, Seth S. Bacterial and fungal study of 100 cases of Chronic Suppurative Otitis Media. J Clin Diagn Res 2011;5:1224-7

22. Chang J, Lee SH, Choi J, Im GJ, Jung HH. Nasopharynx as a microbiologic reservoir in chronic suppurative otitis media: Preliminary study. Clin Exp Otorhinolaryngol 2011;4:122-5.

23. Goycoolea MV, Hueb MM, Ruah C. Otitis media: The pathogenesis approach. Definitions and terminology. Otolaryngol Clin North Am 1991;24:757-61

24. R.R. Marsh, L.W. Tom, Ototoxicity of antimycotics, Otolaryngol Head Neck Surg 100 (1989) 134—136.

25. L.W. Tom, Ototoxicity of common topical antimycotic preparations, Laryngoscope 110 (2000) 509—516.

26. P.J. Damato, Therapy with tolnaftate in otomycosis due to Aspergillus niger, Minerva Med64 (1973) 24—26.

27. A. del Palacio, M.S. Cuetara, M.J. Lopez-Suso, E. Amor, M. Garau, Randomized prospective comparative study: shortterm treatment with ciclopiroxolamine (cream and solution) versus boric acid in the treatment of otomycosis, Mycoses 45 (2002) 317—328.

28. K.M. Than, K.S. Naing, M. Min, Otomycosis in Burma,

29. H. Yamaguchi, K. Okamoto, M. Shooji, M. Morimatsu, S. Hirai, A case of adult type sialidosis with partial betagalactosidase deficiency without myoclonus, Rinsho Shinkeigaku 23 (1983) 1—8.

30. S. Piantoni, S. Narne, R. Bottin, A. Solazzo, W. Bianchi, 1% bifonazole lotion in the therapy of otomycosis, Clin Ter 130 (1989) 23—27.

31. Andral G, Gavarret J. Ann Chim Phys. 1843; 88.

32. Mayer AFJK. Beobachtung von Cysten mit Fadenpilzen aus dem aussern Gehorgange eines Madchens. Arch Anat Physiol. Wissench Med. 1844; 11: 404-8.

33. Galloway DJ. Otomycosis of the Malay Archipelago. J Laryngol. 1903; 64-70.

34. Cheatle A. Otomycosis. J Laryngol Rhinol Otol. 1920; 35: 3-4.

35. Wolf FT. Relation of various fungi to otomycosis. Arch Otolaryngol. 1947; 46: 361-74.

36. English MP. Otomycosis caused by a ringworm fungus. J Laryngol Otol. 1957; 71: 207-8.

37. Lakshmipati G, Murti RB. Otomycosis. J Indian Med Assoc. 1960; 34: 439-41.

38. Agnihotri VP. Aspergilli causing otomycosis. Die Natur. 1961; 702.

39. Gregson AEW, La Touche CJ. Otomycosis: A neglected disease. J Laryngol Otol. 1961a; 75: 45-69.

40. Gregson AEW, La Touche CJ. The significance of mycotic infections in the etiology of otitis externa. J Laryngol Otol. 1961b; 75: 167-70.

41. Ismail HK. Otomycosis. J Laryngol Otol, 1962; 76: 713-21.

42. Yassin A, Mostofa MA, Moawad MK. Fungus infection of the ear. J Laryngol Otol. 1964; 78: 591-602.

43. Damato PJ. Treatment of otomycosis due to A. niger. The St Lukes Hosp Gaz. 1966; 1: 66-88.

44. Beaney GPE, Broughton A. Tropical otomycosis. J Laryngol Otol. 1967; 81: 987-97.

45. McGonigle JJ, Jillson OF. Otomycosis: an entity. Arch Dermatol. 1967; 95: 45-6.

46. Sood VP, Sinha A, Mohapatra LN. Otomycosis: A clinical entity - clinical and experimental study. J Laryngol Otol. 1967; 81: 999-1004.

47. Youssef YA, Abdou MH. Studies on fungus infection of the external ear. I. Mycological and clinical observations. J Laryngol Otol. 1967a; 81: 401-12.

48. Youssef YA, Abdou MH. Studies on fungus infection of the external ear. II. On the chemotherapy of otomycosis. J Laryngol Otol. 1967b; 81: 1005-12.

49. Chandler J R. Malignant external otitis. Laryngoscope. 1968; 78: 1257-94.

50. Yamashita K. [Fungus diseases in otolaryngology, with special reference to otomycosis] Iryo. 1969; 23: 1233-40. Japanese.

51. Bezjak V, Arya OP. Otomycosis due to Aspergillus niger. East Afr Med J. 1970; 47: 247-53.

52. Civila ES, Tost JF, Jimenez AL, Conti-Diaz IA. Root-like hyphal structures in aspergillar otomycosis. Arq Inst Biol (Sao Paulo). 1972; 39: 137-9.

53. Damato PJ. Therapy with tolnaftate in otomycosis due to Aspergillus niger. Minerva Med (Italian). 1973; 64: 24-6.

54. Schonebeck J, Zakrisson JE. Topical 5-fluorocytosine therapy in otomycosis. J Laryngol Otol. 1974; 88: 227-31.

55. Meunier A, Blassin R, Meunier F. Remarquable efficacite due to naftate sur les otomycoses (30 observations). Annales D'otolaryngol (Paris). 1975; 92: 699-702.

56. Kley E. A contribution to the treatment of otomycosis. Laryngol Rhinol Otol. 1976; 55: 765-7.

57. Lawrence TL, Ayers LW, Saunders WH. Drug therapy in otomycosis: an in vitro study. Laryngoscope. 1978; 88: 1755-60.

58. Sengupta RP, Kacker SK. Otomycosis. Indian J Med Sci. 1978; 32: 5-7.

59. Yassin A, Maher A, Moawad MK. Otomycosis : a survey in the eastern province of Saudi Arabia. J Laryngol Otol. 1978; 92: 869-876.

60. Caruso VG. When the patient has otitis externa. Geriatrics. 1980; 35: 35-42.

61. Joy MJ, Agarwal MK, Samant HC. Mycological and bacteriological studies in otomycosis. Indian J Otolaryngol. 1980; 32: 72-5.

62. Chander J, Maini S, Subrahmanyan S, Handa A. Otomicosis—a clinico-medical study and efficacy of

mercurochrome in its treatment. Mycopathologia. 1996;135:9–12.

63. Gunnani HC, Okafor BC, Nzelibe F, Njoku-Obi AN. Etiological agents of otomycosis in Nigeria. Mycoses. 1989;32:224–9.

64. Hennequin C, Bez M, Trotoux J, Simonet M. Otomycose Scopulariopsis brevicaulis apres tympanoplastie. Ann Otolaryngol Chir Cervicofac. 1994;111:353–4.

65. Yasin A, Maher A, Moawad MH. Otomycosis: a survey in the eastern province of Saudi Arabia. J Laryngol Otol. 1978;92:869–76.

66. Jain SK, Agrawal SC. Fungitoxic effect of some organic volatile substances against fungi causing otomycosis.Mycoses. 1994;37:299–301.

67. Ma´rquez A, Garcı´a-Martos P, Marı´n P, Delgado D, Garcı´a Cantos MD, Mira J. Otomicosis de etiologı´a poco comu´n. Enferm Infecc Microbiol Clin. 1999;17:243–5.

68. Baumgartner BJ, Rakita RM, Backous DD. Scedosporium apiospermum otomycosis. Am J Otoryngol. 2007;28:254–6.

69. Aneja KR, Sharma C, Joshi R. Fungal infection of the ear; a common problem in the north eastern part of Hayana. Int J Pediatr Otorhinolaryngol. 2010;74:604–7

70. Pontes ZB, Silva AD, Lima Ede O, Guerra Mde H, Oliveira NM, Carvalho Mde F, Guerra FS. Otomycosis: a retrospective study. Braz J Otorhinolaryngol. 2009;75:367–70.

71. Mohanty JC, Mohanty SK, Sahoo RC, Ghosh SK et al. Clinico microbial profile of Otomycosis in Berhampur. Indian J of Otology 1999;5(2): 81-83.

72. Chander J, Maini S, Subbrahmanyan S and Handa A.Otomycosis-A Cinicomycological study and Efficiency of Mercurochrome in its Treatment. Mycopathologia 1996;135(1): 9-12.

73. Paulose KO,Al khalifa S,ShenoyP and Sharma RK. Mycotic Infection of the ear(Otomycosis) A prospective study. The J of Laryngology and Otology 1989; 103:30- 35.

74. Kaur R,Mittal N,Kakkar M,Aggarwal A, Mathur K. Otomycosis a clinicomycologic study. Ear Nose Throat J 2000;79: 606-609.

75. Sterm JE, Lucente FE. Otomycosis. Ear Nose Throat J.1988;67:804–810.

76. Ibekwe AC, Okafor JI. Pathogenic organisms in chronic suppurative otitis media in Enugu, Nigeria. Trop Geogr Med 1983; 35:389–391

77. Thom C, Church MB. The aspergilli. Baltimore: Williams and Wilkins; 1926.

78. Mitra AK. Otomycosis in a woman caused by Aspergillus niger Tieghem. Curr Sci 1940; 9:371–373.

79. Loh KS, Tan KK, Kumarasinghe G, Leong HK, Yeoh KH. Otitis externa: the clinical pattern in a tertiary institution in Singapore. Ann Acad Med Singapore 1998; 27:215–218

80. Proctor B. The development of the middle ear spaces and their surgical significance. J Laryngol Otol 1964;78:631–648

81. Wigand ME, Trillsch K. Surgical anatomy of the sinus epitympani. Ann Otol Rhinol Laryngol 1973;82:378–383.

82. J. Chander, S. Maini, S. Subrahmanyan, and A. Handa, “Otomycosisa—a clinico-mycological study and efficacy of mercurochrome in its treatment,” Mycopathologia, vol. 135, no. 1, pp. 9–12, 1996.

83. J. C. Mohanty, S. K. Mohanty, R. C. Sahoo et al., “Clinico-microbial profile of otomycosis in Berhampur,” Indian Journal of Otology, vol. 5, no. 2, pp. 81–83, 1999

84. S. R. Agrawal, A. K. Jain, R. B. Goyal, A. Gupta, and K. G. Gupta, “A clinicomycological study of otomycosis with special reference to silent tympanic membrane perforation,” Indian Journal of Otology, vol. 7, no. 2, pp. 49–52, 2001.

85. A. Kumar, “Fungal spectrum in otomycosis patients,” JK Science, vol. 7, no. 3, pp. 152–155, 2005.

86. Z. B. V. D. S. Pontes, A. D. F. Silva, E. D. O. Lima et al., “Otomycosis: a retrospective study,” Brazilian Journal of Otorhinolaryngology, vol. 75, no. 3, pp. 367–370, 2009.

87. P. Kurnatowski and A. Filipiak, “Otomycosis: prevalence, clinical symptoms, therapeutic

procedure,”Mycoses, vol. 44, no. 11-12, pp. 472–479, 2001

88. Machado O, Romeo M. Um caso raro de otomicose bilateral por Hormodendrum. Rev Flumin Med. 1940;5:249-57.

89. . Magalhães O. Ensaios de micologia. Contribuição ao conhecimento dos cogumelos patogênicos em Minas Gerais. Mem Inst Oswaldo Cruz. 1945;42:41-55.

90. Cavalcanti W. Otomycose. Brasil-med. 1941;55:726-9.

91. ***Shelton, J. C., P. J. Antonelli, and R. Hackett.*** *2002. Skull base fungal osteomyelitis in an immunocompetent host. Otolaryngol. Head Neck Surg. 126:76-78.*

ANNEXURE

ABBREVIATIONS

CSOM –CHRONIC SUPPURATIVE OTITIS MEDIA

SDA-SABOURAUD'S DEXTROSE AGAR

TM-TYMPANIC MEMBRANE

KOH-POTASSIUM HYDROXIDE

ET –Eustachian Tube

GERD- Gastroesophageal reflux disease.

MASTER CHART

Sl. No.	AG gp	SEX	ED	HL	ITC	PN	EB	Mic	KOH	FC	F1	F2	F3	RESULT
1	2	M	1	2	1	1	2	1	1	2	2	2	2	2
2	6	M	1	1	2	1	2	1	1	1	1	1	2	1
3	5	F	1	2	1	1	2	1	1	1	1	2	2	1
4	6	F	1	2	1	2	1	1	1	1	1	2	2	1
5	4	M	1	2	1	1	2	1	1	2	2	2	2	2
6	6	F	1	2	1	1	2	1	2	1	1	2	2	1
7	2	M	1	2	2	1	1	1	2	2	2	2	2	2
8	3	F	1	1	1	1	1	1	2	1	1	1	1	1
9	2	F	1	2	1	1	2	1	1	1	1	2	2	1
10	3	F	1	1	1	1	2	1	2	2	2	2	2	2
11	2	F	1	2	1	1	2	1	2	1	1	2	2	1
12	5	F	1	1	1	1	1	1	2	1	1	1	2	1
13	8	M	1	1	1	2	1	1	2	1	2	1	2	1
14	5	F	1	2	1	2	1	1	2	2	2	2	2	2
15	1	M	2	1	1	1	1	1	1	1	2	1	2	1
16	2	M	1	1	1	1	2	1	2	2	2	2	2	2
17	2	M	1	2	1	2	2	1	2	1	2	2	2	2
18	5	M	1	2	2	1	1	1	1	1	2	2	1	1

19	2	F	1	1	1	1	1	1	1	1	1	1	2	1
20	2	F	1	1	2	2	1	1	1	1	1	2	2	1
21	6	M	1	2	1	2	1	2	2	1	2	2	2	2
22	2	F	1	2	1	1	1	1	1	1	1	2	1	1
23	4	M	1	2	1	1	2	1	2	2	2	2	2	2
24	10	M	1	2	2	1	2	1	2	1	2	2	2	2
25	3	F	1	1	2	1	2	1	2	2	2	2	2	2
26	4	M	1	2	2	1	2	1	2	2	2	2	2	2
27	3	M	1	1	1	1	1	1	1	1	1	2	2	1
28	8	F	1	2	1	1	2	1	1	1	1	1	2	1
29	6	F	1	1	2	1	1	1	1	1	2	1	1	1
30	4	F	1	2	1	1	1	1	2	1	1	2	2	1
31	4	M	2	2	1	1	2	1	2	1	1	2	2	1
32	3	F	1	1	2	1	1	1	2	2	2	2	2	2
33	3	F	1	2	1	1	1	1	1	1	2	1	2	1
34	6	M	2	1	2	2	1	1	2	2	2	2	2	2
35	4	F	1	1	1	1	1	1	1	1	1	2	2	1
36	9	F	1	2	1	1	2	1	1	1	1	1	2	1
37	5	M	1	2	1	1	1	1	1	2	2	2	2	2
38	4	F	1	2	1	2	1	1	1	1	1	1	2	1
39	3	M	1	1	1	2	1	1	1	1	2	1	2	1

40	3	M	2	1	1	1	1	1	1	1	2	2	2	2
41	7	F	1	1	1	2	1	1	1	1	2	1	2	1
42	1	M	1	1	1	1	2	1	1	1	1	2	1	1
43	6	M	2	1	1	1	2	1	1	1	1	2	1	1
44	6	M	1	2	1	1	1	1	1	1	2	2	2	2
45	3	M	1	2	1	1	2	1	1	1	2	2	2	2
46	6	F	1	1	1	1	2	1	1	1	2	1	2	1
47	2	F	1	1	1	1	1	1	1	1	1	1	2	1
48	9	F	1	1	1	1	1	1	1	1	1	1	2	1
49	4	M	2	1	1	2	2	1	1	2	2	2	2	2
50	3	F	1	1	1	1	1	1	1	1	1	1	2	1
51	1	F	1	1	1	1	2	1	1	1	1	1	2	1
52	2	F	1	1	1	1	1	1	1	2	2	2	2	2
53	2	M	1	1	1	1	1	1	1	1	2	1	2	1
54	4	M	1	1	1	2	1	1	1	1	1	1	2	1
55	5	M	1	2	1	1	1	1	1	1	1	1	2	1
56	5	M	1	1	1	1	1	1	1	1	1	2	2	1
57	5	M	1	2	1	1	1	1	1	1	1	2	2	1
58	4	M	1	1	1	1	1	1	1	1	1	2	2	1
59	9	F	1	1	1	2	1	1	1	1	1	1	2	1
60	3	F	1	1	1	1	1	1	1	2	1	2	2	1

61	3	M	1	1	1	2	1	1	1	2	2	2	2	2
62	4	F	1	1	2	1	2	1	1	1	1	2	2	1
63	3	M	1	1	1	1	2	1	1	1	2	1	2	1
64	2	F	1	1	1	1	2	1	1	2	2	2	2	2
65	5	F	2	1	1	2	2	1	1	2	2	2	2	2
66	8	F	1	1	2	2	2	1	1	1	2	2	1	1
67	5	F	1	2	1	2	2	1	1	1	1	2	2	1
68	5	F	1	2	1	2	2	1	1	1	2	2	2	2
69	3	F	1	2	2	1	1	1	1	1	2	1	2	1
70	2	F	1	1	1	1	1	1	1	1	1	2	2	1
71	8	F	1	1	1	2	1	1	1	1	2	1	2	1
72	6	F	1	1	2	2	1	1	1	1	1	2	2	1
73	2	F	1	1	2	1	1	1	1	1	2	2	2	2
74	4	F	1	2	1	2	2	1	1	1	1	1	2	1
75	4	F	1	1	1	2	1	1	1	1	1	1	2	1
76	7	F	1	1	1	2	1	1	1	1	1	2	2	1
77	3	M	1	1	1	1	1	1	1	1	1	2	2	1
78	5	M	2	1	1	1	1	1	1	2	2	2	2	2
79	3	M	2	2	1	2	1	1	1	2	2	2	2	2
80	2	M	1	1	2	1	1	1	1	1	2	1	2	1
81	5	F	2	1	1	1	2	1	1	1	2	1	2	1

82	6	F	1	1	1	1	1	1	1	1	1	2	2	1
83	5	M	1	1	1	1	1	1	1	1	2	2	1	1
84	4	M	1	1	2	2	2	1	1	1	2	2	2	2
85	2	M	1	1	1	2	2	1	1	1	2	1	2	1
86	2	F	1	1	2	1	1	1	1	1	1	1	2	1
87	1	F	1	1	1	2	2	1	1	1	1	1	2	1
88	4	M	1	1	2	1	1	1	1	2	2	2	2	2
89	5	M	1	1	1	1	1	1	1	1	2	2	1	1
90	3	F	2	1	1	1	2	1	1	1	2	2	2	2
91	2	M	2	1	2	1	1	1	1	1	1	1	2	1
92	3	F	1	1	1	1	1	1	1	1	1	2	2	1
93	4	F	1	1	1	1	1	1	1	1	1	1	2	1
94	5	M	1	2	1	1	1	1	1	1	2	1	2	1
95	5	M	1	1	1	2	2	1	1	1	1	1	2	1
96	1	M	1	2	2	1	2	1	1	1	1	2	2	1
97	3	F	1	2	2	1	2	1	1	1	2	1	2	1
98	4	F	1	2	2	1	1	1	1	2	2	2	2	2
99	4	M	1	1	1	1	1	1	1	1	2	1	2	1
100	3	F	1	1	2	1	1	1	1	2	2	2	2	2

CONSENT FORM

INFORMED CONSENT FORMAT FOR RESEARCH PROJECTS

I Dr. Arijit Das, am carrying out a study on the topic: INCIDENCE AND TYPES OF OTOMYCOSIS IN CHRONIC SUPPURATIVE OTITIS MEDIA IN PSGIMS& R.as part of my / our research project being carried out under the aegis of the Department of ENT

(*Applicable to students only*): My / our research guide is:

Dr. S. Palaninathan.

Sample size: 100.

Location:_PSGIMS & R.

We request you to kindly cooperate with us in this study. We propose collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration):___20_____ minutes.

Clinical examination (Specify details and purpose): Using otoscope or endoscope to diagnose and take samples if required to effectively treat otomycosis and CSOM

All data and patient details in this study collected in this study will be kept highly confidential.

Benefits from this study: Identification of and typing of otomycosis causing fungi in patients and general population of Coimbatore leading to better treatment

Risks involved by participating in this study: nil

How the results will be used: Reference for understanding pattern of OTOMYCOSIS , its prime causes and following treatment to be given

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, you have the right to withdraw from the interview / study at anytime. You have the freedom to withdraw from the study at any point of time. Kindly be assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will NOT be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of

the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, – whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator/s. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date:

Witness:

PROFORMA

Name: Hospital no.

Age/Sex: IP no.

Address: Date:

Clinical data

Symptoms:

Ear discharge:

- onset , duration, progressive
- Appearance, colour
- Precipitating factors if any (swimming, probing)
- Blood stained or not
- Operated in past(+/-)

Ear pain:

- Intermittent , Progressive (+/-)
- Associated with cold , headache , nasal symptoms
- History of self probing in ear, itching (+/-)
- Referrred pain

Hearing loss :

- Onset , duration
- Severity , associated nasal complaints
- Tinnitus, giddiness

Nasal symptoms:

Allergy, recurrent nose block

Throat:

Duration, onset and progression

Others: Weight loss , fever, vomiting.

Past history:

Diabetes, Hypertensive, TB ,Asthma , previous surgery, drug intake etc

Personal history

Ocuupation, smoker / alcoholic , socioeconomic status,

Sleep, apptite, bowel bladder habits

Family history

History of similar illness in family members

General Examination:

Built: Anemia ,cyanosis, clubbing , edema, lymphadenopathy

Vital signs: Pulse, Temperature, blood pressure, Respiratory rate

ENT examination:

Ears:

- Pinna, Pre and post auricular areas
- Mastoid/tragal tenderness
- EAC
- TM appearance(when seen)
- Tuning for tests

Facial Nerve function

Nose:

External framework,

Anterior rhinoscopy

Posterior rhinoscopy

Examination of Paranasal sinus tenderness

Throat:

Oral cavity –Abnormality if any of lips , buccal mucosa, hard , soft palate, tonsils.

Oropharynx: congestion, fullness.

Indirect laryngoscopy:

Systemic exam:CNS, CVS, Rspiratory system, Abdominal examination

Routine: CBC, RBS,URINE ROUTINE

Chest X-RAY, Urea/Creatinine (>40 yrs)

OTOENDOSCOPY

EAR SWAB FOR KOH MOUNT , FUNGAL CULTURE

X.RAY Temporal bone(lateral oblique view) (If planned for surgery)

Treatment: Medical and/or Surgical

Follow up: Improvement in symptoms